DYSLIPIDEMIA AND HYPERTENSION IN OBESE PATIENTS WITH CORRELATION TO BODY MASS INDEX

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CERTIFICATE

This is to certify that this dissertation titled **"DYSLIPIDEMIA AND HYPERTENSION IN OBESE PATIENTS WITH CORRELATION TO BMI "** submitted by **DR.N.SURESH** to the faculty of General Medicine, **The Tamil Nadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the requirement for the award of MD degree branch I General Medicine, is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I **Dr.N.SURESH** ; solemnly declare that the dissertation titled **"DYSLIPIDEMIA AND HYPERTENSION IN OBESE PATIENTS WITH CORRELATION TO BMI"** has been prepared by me.

This is submitted to **The Tamil Nadu Dr.M.G.R. Medical University, Chennai,** in partial fulfillment of the requirement for the award of MD degree (branch I) General Medicine.

Place: Madurai.

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CONTENTS

INTRODUCTION

Obesity is a chronic and increasingly common health hazard characterized by excess body fat. It develops gradually and often persists throughout life. As a preventable cause of death in the US, obesity is second only to smoking.

Traditionally, obesity was believed to be associated with affluent lifestyles in the West countries. However, obesity is a fast growing problem in developing countries and is now known to be associated with increased health risks. Several studies in India have shown that changes in dietary patterns, physical activity levels, life styles associated with affluence and migration to urban areas are related to increasing risk of metabolic diseases such as type 2 diabetes mellitus, hypertension and dyslipidemia.

The most widely used method to gauge obesity is the Body mass index (BMI) and it focus in obesity treatment recommendations, with different treatment cutoff points based up on the presence or absence of obesity-related comorbid disease. In addition, many patients with these metabolic diseases are either overweight or obese. Important relationship

between BMI and metabolic disease is critical toward a better understanding of the underlying pathophysiological processes leading to excessive fat-related metabolic disease.

This study brings out the important risk factors for cardiovascular disease like hypertension and dyslipidemia in obese patients correlated with various BMI levels.

REVIEW OF LITERATURE

OBESITY

Prevalence of Obesity

The worldwide prevalence of obesity has increased dramatically over the last several decades. In the United States alone, about one third of adults aged 20 to 74 years are considered obese.[1] According to national population surveys conducted since 1960, the prevalence of obesity (BMI >30 kg/m^2) has more than doubled from 13% to 32% [1] [2]; the prevalence of obesity increases progressively from 20 to 50 years of age, but it then declines after 60 to 70 years of age.

The prevalence of obesity has also risen in children and adolescents. As defined by a BMI greater than the 95th percentile for age and gender from the revised National Center for Health Statistics growth charts, 17% of 6- to 17-year-old children and adolescents in the United States are overweight [1] [3]. These data indicate that overweight prevalence rates for children and adolescents, reported by earlier surveys, have doubled. Diseases commonly associated with obesity in adults, such as type 2 diabetes mellitus, hypertension, hyperlipidemia, gallbladder disease, nonalcoholic steatohepatitis (NASH), sleep apnea, and orthopedic complications, are now increasingly observed in children.

INDIAN SCENARIO

Obesity is emerging as an important problem in India. Twenty two million Indians are obese, especially abdominally obese. The Nutrition Foundation of India (NFI) study showed that 32.3% of middle class males and 50% of middle class females in Delhi are obese[4].

Prevalence pattern of obesity across different age groups studied rural setting in Kerala included 3423 subjects over 20 years of age showed 30-40 % of the population in their study setting was either obese or overweight[5].

Prevalence of obesity in adults--an epidemiological study from Kashmir Valley of India showed that according to BMI, the overall prevalence of obesity in the study population was 15.01%; The prevalence of obesity in males was 7.01% and in females 23.69%[6].

Prevalence of overweight in urban Indian adolescent school children study showed that Age-adjusted prevalence of overweight was 17.8% for boys and 15.8% for girls[7].

Definition of Obesity

Obesity is a state of excess adipose tissue mass. Although often viewed as equivalent to increased body weight, this need not be the case -

-lean but very muscular individuals may be overweight by arbitrary standards without having increased adiposity.[8]

Assessing Obesity

Visual inspection of a patient can give a subjective but fairly accurate estimate of the degree of obesity. Obesity is usually easily diagnosed using what has been called the eyeball test. 'If a person looks fat, the person is fat.'

Presently, there are three commonly used objective methods of estimating obesity in clinical practice: body mass index, waist-to-hip ratio and waist circumference and fat distribution[4].

Body Mass Index

The most widely used method to gauge obesity is the Body Mass Index. (Also called Quetelet Index)

Body Mass Index (BMI) = weight/height^2 (in kg/m^2)

Most authorities use the term overweight (rather than obese) to describe individuals with BMIs between 25 and 30. A BMI between 25 and 30 should be viewed as medically significant and worthy of therapeutic intervention, especially in the presence of risk factors that are influenced by adiposity, such as hypertension and glucose intolerance.

Large epidemiologic studies[9,10] established an inverse relationship between BMI and mortality above BMI values of 25.0 kg/m^2. Men and women with a BMI between 25.0 and 29.9 kg/m^2 are considered overweight, and those with a BMI greater than 30.0 kg/m^2 are considered obese. Obese persons are at higher risk for adverse health consequences than those who are overweight. These criteria for overweight and obesity represent imposed cutoff values along a continuum between mortality rate and BMI.

Classification of Weight Status and Risk of Disease[8]

Table :1

	BMI (kg/m^2)	**Obesity Class**	**Risk of Disease**
Underweight	<18.5		
Healthy weight	18.5–24.9		
Overweight	25.0–29.9		Increased
Obesity	30.0–34.9	I	High
Obesity	35.0–39.9	II	Very high
Extreme Obesity	≥ 40	III	Extremely high

Source: Adapted from National Institutes of Health, National Heart, Lung, and Blood Institute: Clinical Guidelines on the Identification,

Evaluation, and Treatment of Overweight and Obesity in Adults. U.S. Department of Health and Human Services, Public Health Service, 1998.

The BMI for native Asian Indians [4]

Table :2

Classification of Overweight and obesity by Body mass index (BMI) for Indians		
	Obesity Class	**BMI(kg/m^2)**
Under weight		<18.5
Normal		18.5-22.9
Overweight		23.0-27.9
Obesity	I	28.0-32.9
	II	33.0-37.9
Extreme obesity	III	≥38

Waist-to-Hip Ratio (WHR) and Waist Circumference

The waist-to-hip ratio provides information about the distribution of body fat

Body Fat Distribution (Fat Phenotypes)

On the basis of distribution of body fat, obesity may be classified in to android obesity and gynoid obesity.

Apple type and pear shaped obesity

On the basis of body fat distribution, obesity may be classified into android and gynoid types.

Android obesity is the collection of fat mostly in the abdomen (above the waist). It is also called central obesity and is associated with an increased risk of metabolic complications such as CAD, DM, HT and dyslipidemia. It is the most common acquired cause of insulin resistance. Also known as apple type body.

Gynoid obesity (pear shaped body) is the collections of fat on the hips and buttock (below the waist or gluteo femoral). This makes the person more prone to mechanical disorders such as varicose vein and disorders of the joints.

Measurement of central obesity

Waist hip ratio (WHR)

The usual measure of central obesity is waist hip ratio. Since the excess fat is usually concentrated in the hip in women and the waist in men, the optimum value for the waist hip ratio is lower in women(<0.85)

than in men (<0.95). The desired waist to hip ratio in men is ≤ 1.0 and in women is ≤ 0.8[4].

Waist hip ratio > 1.0 in men and >0.9 in women is abnormal (Harrison p.463) and is associated with high morbidity and mortality.

Waist circumference

Recently waist circumference has been found to be even better market of central obesity than waist hip ratio. For Asian Indians the optimum waist circumference is <80cm in women and <90cm in men[11].

In men there is increased risk if the waist circumference is 85cm or more and substantial risk if it is 90cm or more. For women the figures are ≥ 80 and ≥ 85cms respectively[4].

Many of the most important complications of obesity, such as insulin resistance, diabetes, hypertension, hyperlipidemia, and hyper androgenism in women, are linked more strongly to intra abdominal and/or upper body fat than to overall adiposity .The mechanism underlying this association is unknown but may relate to the fact that intra abdominal adipocytes are more lipolytically active than those from other depots. Release of free fatty acids into the portal circulation has adverse metabolic actions, especially on the liver.

BMI-associated health risk is also influenced by ethnicity[12], the risk of diabetes is higher in Southeast Asian populations than in whites.

Another factor that modifies the risk of obesity-related complications is weight gain during adulthood. In both men and women, weight gain of 5 kg or more since the ages of 18 to 20 years increases the risk of developing diabetes, hypertension, and coronary heart disease, and the risk of disease increases with the amount of weight gained [13,14,15,16].

Etiology of Obesity

Energy Balance

Obesity is caused by an excessive intake of calories in relation to energy expenditure over a long period of time. Large increases in body fat can result from even minor, but chronic, differences between energy intake and energy expenditure. In 1 year, the ingestion of only 5% more calories than expended can promote the gain of approximately 5 kg in adipose tissue. Over 30 years, the ingestion of only 8 kcal /day more than expended can increase body weight by 10 kg[17].

Genes and Environment

Body size depends on the complex interaction between genetic background and environmental factors. In humans, genetic background explains only an estimated 40% of the variance in body mass.[18]

SOME REASONS FOR THE INCREASING PREVALENCE OF OBESITY – THE 'OBESOGENIC' ENVIRONMENT[19]

Increasing energy intake

1. Increasing portion sizes
2. Increasing snacking and loss of regular meals
3. Increasing energy-dense food (mainly fat)
4. Increasing affluence

Decreasing energy expenditure

1. Increasing car ownership
2. Decreasing walking to school/work
3. Increasing automation; decreasing manual labour.
4. Decreasing sports in schools
5. Increasing time spent on video games and watching TV
6. Increasing Central heating

Influences of Childhood and Parental Obesity

The risk of becoming an obese adult is influenced both by having been obese as a child and by having had at least one obese parent..The risk of being obese at 21 to 29 years of age ranged from 8% for persons who were obese at 1 to 2 years of age and had nonobese parents to 79% for persons who were obese at 10 to 14 years of age and had at least one obese parent.[20]

Obesity Genes in Humans

Table:3

Gene	Gene Product	Mechanism of Obesity
Lep (*ob*)	Leptin, a fat-derived hormone	Mutation prevents leptin from delivering satiety signal; brain perceives starvation
LepR (*db*)	Leptin receptor	Same as above
POMC	Proopiomelanocortin, a precursor of several hormones and neuropeptides	Mutation prevents synthesis of melanocyte-stimulating hormone (MSH), a satiety signal
MC4R	Type 4 receptor for MSH	Mutation prevents reception of satiety signal from MSH
PC-1	Prohormone convertase 1, a processing enzyme	Mutation prevents synthesis of neuropeptide, probably MSH
TrkB	TrkB, a neurotrophin receptor	Hyperphagia due to uncharacterized hypothalamic defect

Syndromes associated with of Obesity [8]

1. Prader-Willi syndrome
2. Laurence-Moon-Biedl syndrome
3. Ahlstrom syndrome
4. Cohen syndrome
5. Carpenter syndrome

POTENTIALLY REVERSIBLE CAUSES OF WEIGHT GAIN[19]

Endocrine factors	**Drug treatments**
Hypothyroidism	Tricyclic antidepressants
Hypothalamic tumours or injury	Corticosteroids
Cushing's syndrome	Sulphonylureas
Insulinoma	Oestrogen-containing contraceptive pill
	Corticosteroids
	Sodium valproate
	β- Blockers

Pathogenesis of Obesity

Energy Metabolism

The components of daily total energy expenditure (TEE) are resting energy expenditure (REE), which accounts for approximately 70% of TEE; energy expended in physical activity, which accounts for approximately 20% of TEE; and the thermic effect of food (TEF), which accounts for approximately 10% of TEE. REE represents the energy expended for normal cellular and organ function under post absorptive resting conditions.

Evidence from studies in obese and lean subjects, matched for either fat mass or lean body mass, suggests that obese subjects have a small (75 kcal/day) but potentially important reduction in TEF. This reduction in TEF might arise from the insulin resistance and blunted sympathetic nervous system activity that occur in obesity.[53]

Obesity causes many serious medical complications that impair quality of life and lead to increased morbidity and premature death[21]

Clinical Features and Complications of Obesity

Endocrine and Metabolic Diseases

The Metabolic or Insulin-Resistance Syndrome

Type 2 Diabetes Mellitus

Dyslipidemia

Cardiovascular Disease

Hypertension

Coronary Heart Disease

Cerebrovascular and Thromboembolic Disease

Pulmonary Disease

Restrictive Lung Disease

Obesity-Hypoventilation Syndrome

Obstructive Sleep Apnea

Musculoskeletal Disease

Gout

Osteoarthritis

Cancer

In both men and women, BMI was also significantly associated with higher rates of death due to cancers of the esophagus, colon and rectum, liver, gallbladder, pancreas, and kidney; non-Hodgkin's lymphoma; and multiple myeloma. Significant trends of increasing risk with higher BMI values were observed for death from cancers of the stomach and prostate in men and for death from cancers of the breast, uterus, cervix, and ovary in women.[22]

Genitourinary Disease in Women

Neurologic Disease

Obesity increases the incidence of ischemic stroke.

Obesity is also associated with idiopathic intracranial hypertension (IIH), also known as pseudo tumor cerebri.

Cataracts

Gastrointestinal Disease

Gastro esophageal Reflux Disease

Gallstones

Pancreatitis

Liver Disease

Obesity is associated with a spectrum of liver abnormalities, which are now referred to as nonalcoholic steatohepatitis.[23]

Metabolic or insulin-resistance syndrome, also known as syndrome X, the specific phenotype of upper-body, or abdominal, obesity is associated with a cluster of metabolic risk factors for coronary heart disease (CHD).

Features of this syndrome include

- Insulin resistance with associated hyperinsulinemia
- Impaired glucose tolerance, impaired insulin-mediated glucose disposal, and type 2 diabetes mellitus;
- Dyslipidemia, characterized by hypertriglyceridemia and low serum HDL-cholesterol levels and Hypertension.

Other metabolic risk factors, including

- Increased serum levels of apolipoprotein B;
- Small, dense LDL particles; and
- Plasminogen activator inhibitor 1 (PAI-1) with impaired fibrinolysis have also been associated with abdominal obesity. (24)(25)

There is increasing evidence that the ectopic distribution of triglycerides in nonadipose tissue may be involved in the complications

of obesity. In cross-sectional studies, insulin resistance was highly correlated with the intramyocellular concentration of triglyceride.[26]

DIAGNOSTIC CRITERIA FOR METABOLIC SYNDROME

Table:4

Clinical measure	**WHO (1998)**	**ATP III (2001)**
Insulin resistance	IGT, IFG or lowered insulin sensitivity plus any two of the following	None, but any 3 of the following 5 features
Body weight	Men: WHR > 0.90 Women : WHR > 0.85 And / or BMI > 30kg/m^2	WC ≥102 cm in men or ≥ 88 cm in women
Lipid	TG >150 mg/dL and /or HDL-C < 35mg/dL in men or < 39mg/dL in women	TG >150mg/dL HDL-C < 40mg/dL in men or < 50mg/dL in women
Blood pressure	> 140/90 mmHg	≥ 130/85 mmHg
Glucose	IGT, IFG or T2DM	> 110 mg/dL (includes diabetes)
Microabluminuria	Urine albumin excretion rate: 20 mcg per minute	Other features of insulin resistance

The criteria by WHO and ATP III for diagnosing metabolic syndrome may not be appropriate for Asians. IDF (International diabetes federation) had made a first attempt to provide ethnic group specific cut off points for some risk factor such as waist circumference.

IDF criteria for metabolic syndrome

Increased waist circumference (population specific) plus any 2 of the following:

- TGL ≥ 150mg/dL or on specific treatment for TGL
- HDL < 40mg/dL in men or < 50mg/dL in women or on treatment for this lipid abnormality.
- BP ≥ 139mm of Hg systolic or ≥ 85mm of Hg diastolic or on antihypertensive treatment.
- Fasting plasma glucose ≥ 100mg/dL (includes diabetes).

Type 2 Diabetes Mellitus in Obesity

The marked increase in the prevalence of obesity has played an important role in the 25% increase in the prevalence of diabetes that has occurred in the United States over the last 20 years[27]. According to data from NHANES III, two thirds of the men and women in the United States with diagnosed type 2 diabetes have a BMI of 27.0 kg/m^2 or greater.[28]. The risk of diabetes increases linearly with BMI: the prevalence of diabetes increased from 2% in those with a BMI of 25.0 to 29.9 kg/m^2, to 8% in those with a BMI of 30 to 34.9 kg/m^2, and to 13% in those with a BMI greater than 35 kg/m^2.

In the Nurses' Health Study, the risk of diabetes began to increase when BMI exceeded the "normal" value of 22 kg/m^2. [27]

The risk of diabetes also increases with weight gain during adulthood.[29,30,31.] Among men and women aged 35 to 60 years, the risk of diabetes was three times greater in those who gained 5 to 10 kg since the age of 18 to 20 years than in those who maintained their weight within 2 kg.

Dyslipidemia in Obesity

Obesity is associated with several serum lipid abnormalities including hypertriglyceridemia, reduced HDL cholesterol levels, and an increased fraction of small, dense LDL particles. [32] This association is especially strong in persons with abdominal obesity. In addition, most studies suggest that serum concentrations of total and LDL cholesterol are elevated in obesity. Data from NHANES III showed that in men, there was a progressive increase in the prevalence of hypercholesterolemia (total blood cholesterol >240 mg dL or 6.21 mmol/L) with increasing BMI.[33] In women, by contrast, the prevalence of hypercholesterolemia was highest at a BMI of 25.0 to 27.0 kg/m^2, and it did not increase further at higher BMI values. The serum lipid abnormalities associated with obesity are important risk factors for CHD.

Hypertension in Obesity

There is a linear relationship between hypertension and BMI. [127] [128] In NHANES III, the age-adjusted prevalence of hypertension (defined as systolic blood pressure >140 mm Hg, diastolic blood pressure >90 mm Hg, or use of antihypertensive medication) in obese men and women was 42% and 38%, respectively. These prevalence rates are more than twice as high as the prevalence rates of hypertension in lean men and women (□15% prevalence rate in both men and women)[33]. The risk of hypertension also increases with weight gain. Among subjects followed in the Framingham Study, there was a 6.5-mm Hg increase in blood pressure with every 10% increase in body weight.[34]

Weight gain, even to levels not considered to be a problem, increases the incidence of hypertension. This was clearly shown in a Nurses' Health Study.[35].Those women, now in midlife, who had gained as little as 5 kg over their weight at age 18 had a 60% higher relative risk of developing hypertension than did those whose weight had not changed more than 2 kg. Those who gained 10 kg or more had a 2.2-fold greater risk. In a cross-sectional study, adults with a BMI >40 had a 6.38 times greater odds ratio for hypertension [36]

Moreover, obesity is accompanied by an increased incidence of hypertension-related outcomes, including stroke [37,] coronary disease [38] heart failure [39], and restrictive cardiomyopathy [40].

Pathophysiology of hypertension in Obesity

a. Fluid volume distribution and systemic hemodynamic changes [8]

Obese patients have a distinct cardiovascular abnormality characterized by increased total and central blood volumes, cardiac output, and left ventricular filling pressure. The elevated cardiac output appears to be required to support the metabolic needs of the excess adipose tissue. Left ventricular filling pressure is often at the upper limits of normal at rest and rises excessively with exercise. As a result of chronic volume overload, eccentric cardiac hypertrophy with cardiac dilatation and ventricular dysfunction may develop.. Pulmonary congestion, peripheral edema, and exercise intolerance may all ensue;

b. Insulin resistance

c. .sympathetic nervous system activity

Sympathetic activation appears to mediate at least part of the obesity-induced sodium retention and hypertension since adrenergic blockade or renal denervation markedly attenuates these changes [41]

d. Sodium – potassium ATP-ase

e. Renin-angiotensin system (RAS) and serum aldosterone levels

f. Increased leptin from selective leptin resistance[41]

Leptin is an adipocyte-derived hormone that acts in the hypothalamus to regulate appetite, energy expenditure and sympathetic nervous system outflow. One of the major mechanisms leading to the development of obesity-induced hypertension appears to be leptin-mediated sympatho-activation. Leptin adversely shifts the renal pressure–natriuresis curve, leading to relative sodium retention.

g. Increased free fatty acids [43]

h. Decreased nitric oxide[44]

i. Increased endothelin-1[45]

j. Obstructive sleep apnea (OSA)[46]

Among the risk factors for OSA, obesity is probably the most important. Significant sleep apnea is present in ≈40% of obese individuals, and ≈70% of OSA patients are obese Possible mechanisms whereby OSA may contribute to hypertension in obese individuals include sympathetic activation, hyperleptinemia, insulin resistance, elevated angiotensin II and aldosterone levels.

From the clinical and therapeutic perspectives, the presence of resistant hypertension and the absence of a nocturnal decrease in blood pressure in obese individuals should prompt the clinician to consider the diagnosis of OSA, especially if clinical symptoms suggestive of OSA (such as poor sleep quality, witnessed apnea, excessive daytime somnolence, and so forth) are also present.

Adipose Tissue and Triglyceride Metabolism

Triglycerides stored within adipose tissue constitute the body's major energy reserve. Triglycerides are a much more compact fuel than glycogen because of the energy density and hydrophobic nature of fat. Triglycerides yield 9.3kcal/g upon oxidation and are compactly stored as oil inside the fat cell, accounting for 85% of adipocyte weight. Glycogen, in contrast, yields only 4.1kcal/g upon oxidation and is stored intracellularly as a gel containing approximately 2 g of water for every 1 g of glycogen.

Adipose tissue is an effective storage mechanism for transportable fuel that allows mobility and survival when food is scarce. During starvation, the duration of survival is determined by the size of the adipose tissue mass

Triglyceride Storage

The major function of adipocytes is the storage of triglycerides for future use as energy substrate. Lipogenesis from glucose makes only a limited contribution to triglyceride storage in the adipocyte.[47]

Most of the triglyceride in adipocytes is derived from chylomicrons and very-low-density lipoprotein (VLDL) triglycerides that originate,

respectively, from dietary and hepatic sources. These plasma triglycerides are hydrolyzed by lipoprotein lipase (LPL), a key regulator of fat cell triglyceride uptake from circulating triglycerides. Lipoprotein lipase is synthesized by adipocytes and transported to the endoluminal surface of endothelial cells. The interaction of LPL with chylomicrons and VLDL releases fatty acids from plasma triglycerides, which are then taken up by local adipocytes. Plasma free fatty acids themselves can also be taken up by adipose tissue, independent of LPL.

Insulin and cortisol are the principal hormones involved in regulation of LPL activity and expression.[48] The activity of LPL within individual tissues is a key factor in partitioning triglycerides among different body tissues. Insulin influences this partitioning through its stimulation of LPL activity in adipose tissue.[49] Insulin also promotes triglyceride storage in adipocytes through other mechanisms, including inhibition of lipolysis, stimulation of adipocyte differentiation, and escalation of glucose uptake. Cortisol's importance in fat distribution is supported by the clinical appearance of patients with Cushing's syndrome. The obesity-promoting effect of cortisol can involve a synergistic effect of cortisol and insulin on the induction of LPL in adipose tissue, as has been demonstrated in vitro. Testosterone, growth hormone,

catecholamines, tumor necrosis factor (TNF), and other related cytokines inhibit lipoprotein lipase activity.[48]

Adipose Tissue as an Endocrine Organ

Traditionally, adipocytes have been viewed as energy depots that store triglycerides during feeding and release fatty acids during fasting to provide fuel for other tissues. However, adipose tissue secretes numerous proteins that have important physiologic functions. These factors participate in autocrine and paracrine regulation within adipose tissue and can affect the functions of distant organs, such as muscle, the pancreas, the liver, and the central nervous system.

ADIPOCYTE-SECRETED PROTEINS

Table :5

Category	**Protein**
Hormone	Leptin, resistin, angiotensinogen, ACRP 30, estrogens, visfatin
Cytokine	Interleukin-6, tumor necrosis factor-α
Extracellular matrix protein	Types I, III, IV, and VI collagen; fibronectin, osteonectin, laminin, entactin, MMP-2
Complement factor	Adipsin, complement C3, factor B
Enzyme	Cholester ester transfer protein, lipoprotein lipase
Acute phase response protein	α-1 Acid glycoprotein, haptoglobin
Other	Fatty acids, plasminogen activator inhibitor-1, prostacyclin

Leptin

Adipocytes produce leptin and secrete it into the bloodstream. Leptin has pleiotropic effects on food intake, hypothalamic

neuroendocrine regulation, reproductive function, and energy expenditure [50] [51]. There is a direct relationship between plasma leptin concentrations and BMI or body fat percentage

Resistin

Resistin is another signaling polypeptide secreted by adipocytes.[52]

Adiponectin

Adiponectin is the most abundant secretory protein produced by adipocytes. In contrast to other secretory products of adipocytes, the plasma concentrations of adiponectin are decreased in obesity and insulin resistance. There is a close association between hypoadiponectinemia, insulin resistance, and hyperinsulinemia.[53] Conversely, adiponectin expression increases with improved insulin sensitivity and weight loss.[54]

AIMS AND OBJECTIVES

1. To study the prevalence of dyslipidemia, hypertension and diabetes among the obese .

2. To study the dyslipidemia in correlation with Body Mass Index and compare among the different groups.

3. To study about the Hypertension in correlation with Body Mass Index and compare among the different groups.

MATERIALS AND METHODS

Setting **:** Outpatient Department and Medical Wards, Govt Rajaji Hospital, Madurai.

Collaborating Department **:** Department of Biochemistry, Madurai Medical College, Madurai**.**

Design of the study **:** Cross sectional study

Period of the Study **:** JUNE 2007 –MAY 2008

Sample size : 80 patients study group and 20 patients control group.

Ethical approval : Obtained

Consent : Informed consent was obtained

Financial Support : Nil

Conflict of Interest : Nil

SELECTION OF THE STUDY SUBJECTS AND CONTROLS

Among patients attending medical Outpatient Department and Medical Wards in Govt Rajaji Hospital for varying illness , 80 Patients (40 Males and 40 Females) whose BMI were ≥ 25 selected for the study Group(Group II ,III ,IV) and 20 Patients (9 males and 11 Females) whose BMI < 25 were selected in the same age group as controls. (Group I)

Divided into four groups according to BMI

Group	BMI	
I	□25	**Healthy weight**
II	≥ 25 – 26.9	**Overweight**
III	≥27 – 29.9	**Overweight**
IV	≥30	**Obesity**

INCLUSION CRITERIA

Both males and females more than 20 years of age including hypertension and Diabetes Mellitus.

EXCLUSION CRITERIA

1. Less than 20 years of age
2. Pregnancy.
3. Secondary causes affecting the lipid profile was not included

Nephrotic syndrome

Hypothyroidism

Chronic renal failure

Connective tissue disorder

Patients on hypolipidemic drugs, diuretics, β blockers

Patient with previous history of CAD on hypolipidemic drugs

80 patients both males and females more than 20 years of age were taken as cases. 20 age matched males and females were taken as controls.

History

Careful history was first taken in these patients regarding pre existing diabetes mellitus, systemic hypertension and dyslipidemia and about the treatment for these illnesses.

Anthropometry

In all these patients the following anthropometric measurements are done

1. Height
2. Weight
3. Waist Circumference
4. Hip Circumference

From these measurements Body Mass Index and waist hip ratio were calculated.

Blood pressure recording

BP was taken three times to confirm systemic hypertension.

Investigations

The following Investigations were done after a overnight fasting for more than 8 hours,

1. Fasting Plasma Glucose
2. Lipid profile

By history, clinical examination and biochemical analysis, secondary causes of elevated lipid profile was excluded. The collected data's were analyses for correlation with Body Mass index.

DEFINITION

Hypertension – Patient was considered to be hypertensive if patient was diagnosed earlier or if patients was on anti hypertensive medication or if blood pressure during recording it was found to be more than 140mm Hg systolic or 90 mm Hg diastolic according to JNC VII definition.

Diabetes – Patient was considered to be diabetic if she was diagnosed earlier or was on anti diabetic treatment or on admission, found to have fasting plasma glucose more than 126mg% according to ADA Criteria.

Dyslipidemia

Defined to include any one of the following:

Total cholesterol (TC) □ 240 mg/dl

Triglycerides (TG) > 200 mg/dl

Low-density lipoprotein cholesterol (LDL-C) □160 mg/dl

High-density lipoprotein cholesterol (HDL-C) < 40 mg/dl

Height – Height was taken by asking the patient to stand erect without foot wears against to the wall and the vertical height was measured in centimeters.

Weight – Weight was measured in kilograms using a weighing machine.

Body Mass Index

It is calculated by,

Body Mass Index (BMI) = weight/height^2 (in kg/m^2)

Waist circumference – Abdominal girth at the level of equidistant between costal margin and iliac crest ≥ 85cm is taken as abnormal in women and ≥90 cm in Men.

Hip circumference – Body girth at the level of greater trochanter is measured.

Waist hip ratio – is the ratio calculated by dividing waist circumference by hip circumference. Value ≥ 0.85 is taken as abnormal in women and ≥0.95 in Men..

Lipid profile consists of total cholesterol, TG, LDL, HDL and VLDL done direct enzymatic assay.

STATISTICAL ANALYSIS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2002).**

Using this software, range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS AND OBSERVATIONS

PROFILE OF CASES STUDIED

Table 6 : Age distribution

Age group	Test Group		Control Group	
	No	**%**	**No**	**%**
Upto 30 yrs	13	16.3	4	20
31-40	21	26.3	5	25
41-50	19	23.8	5	25
51-60	12	15	5	25
Above 60	15	18.8	1	5
Total	80	100	20	100
Range	21-73		26-72	
Mean	45.0		41.8	
SD	13.5		11.7	
'p'	0.3494 Not Significant			

Mean Age in study group were 45.0 with SD of 13.5 years. Control group mean was 41.8 with SD of 11.7 years. 'p' value 0.3494. The difference was statically not significant. That is the two groups were comparable.

Table 7 : Sex Distribution

Sex	Test Group		Control Group	
	No	**%**	**No**	**%**
Male	40	50	9	45
Female	40	50	11	55
Total	80	100	20	100
'p'	0.8807 Not significant			

Total no of patients - 100

Male - 49

Female - 51

Both sexes were equally distributed in this study and control Groups.

The 'p' value is (0.8807), statistically not significant.

Table 8 : BMI

BMI Group	Test Group		Control Group	
	No	**%**	**No**	**%**
I (<25)	-	-	20	100
II (25 – 26.9)	31	38.8	-	-
III (27 – 29.9)	28	35	-	-
IV (>30)	21	26.3	-	-
Range	25.1-40.3		19.4-24.7	
Mean	28.7		22.3	
SD	3.4		1.6	
'p'	**0.0001** **Significant**			

The mean BMI in Study group were 28.7 with SD of 3.4. Control group mean BMI was 22.3 with SD of 1.6. 'p' value 0.0001. The difference was statistically significant.

Table 9 : BMI and Age

BMI Group	Age Group									
	Upto 30		31 - 40		41-50		51-60		61-70	
	No	%	No	%	No	%	No	%	No	%
I (20)	4	20	5	25	5	25	5	25	1	5
II (31)	7	22.6	7	22.6	6	19.4	5	16.1	6	19.4
III (28)	3	10.7	8	28.6	7	25	4	14.3	6	21.4
IV (21)	3	14.3	6	28.6	6	28.6	3	14.3	3	14.3
Mean BMI	26.3		27.8		28.1		27		27.5	
SD	3.6		4.4		4.5		4.2		2.7	
'p'	0.729 Not Significant									

Total no of patients 100. Mean BMI in age up to 30 years were 26.3 with SD of 3.6. Mean BMI in age group 31- 40 years were 27.8 with SD of 4.4.

Mean BMI in age group 41 -50 years were 28.1 with SD of 4.5.

Mean BMI in age group 51 - 60 years were 27 with SD of 4.2.

Mean BMI in age group 61 - 70 years were 27.5 with SD of 2.7.

'p' value 0.729. Statistically not significant.

Table 10 : BMI and Systolic B.P.

BMI Group	Systolic B.P	
	Mean	**SD**
I	115.0	16.6
II	125	18.6
III	129.3	23.1
IV	134.9	29.8
'p'	**0.0434** **Significant**	

Mean systolic Blood pressure was composed among the difference groups.

Mean Systolic BP in BMI group I (control group) were 115.0 with SD of 16.6.

Mean Systolic BP was increased from Group I to Group IV, which is statistically significant. 'p' value 0.0434.

Table 11 : BMI and Diastolic B.P.

BMI Group	Diastolic B.P	
	Mean	**SD**
I	71.9	11.8
II	76.3	15.5
III	79.1	17.3
IV	84.6	16.5
'p'	**0.0423** **Significant**	

Mean Diastolic Blood pressure is composed among the difference groups. Mean Diastolic BP in BMI group I (control group) were 71.9 with SD of 11.8.

Mean Diastolic BP was increased from Group I to Group IV, which is statistically significant 'p' value 0.0423.

Table 12 .BMI AND HYPERTESION

BMI Group	Hypertension			
	Present		Absent	
	No	%	No	%
I (0)	3	15	17	85
II (31)	9	29	22	71
III (28)	8	28.6	20	71.4
IV (21)	8	38.1	13	61.9
Mean BMI	29.06		25.01	
SD	4.98		3.4	
'p'	0.03151 Significant			

Mean BMI among the Hypertensive patients were 29.06 with SD of 4.98 and Mean BMI among the non hypertensive patients were 25.01 with SD of 3.4. The percentage of Hypertension increased from group I to IV (15 to 38.1 %).

Table 13 : BMI and Lipid Profile

BMI Group	TGL		VLDL		HDL		TC		LDL	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
I (20)	138.2	38	27.6	7.6	54.2	9.1	192.2	27.8	110.4	27.4
II (31)	147.3	36.6	29.5	7.3	49.3	9.3	210.7	27.3	131.9	27.4
III (28)	156.8	41.3	31.4	8.3	48.3	10.5	214.4	27.3	134.8	25
IV (21)	162.5	40.4	32.5	8.1	44.7	10.1	214	30.4	136.8	26.5
'p'	**0.0251 Significant**		**0.0251 Significant**		**0.0208 Significant**		**0.0217 Significant**		**0.0022 Significant**	

TC

The mean total cholesterol in the control group (I) was 192.2 with SD of 27.8. The mean total cholesterol was increased from group I to group III. In Group IV there was not much difference in mean TC with group III. 'p' value 0.0217. The difference was statistically significant.

TGL

The mean TGL (triglyceride) in the control group (I) was 138.2 with SD of 38. The mean TGL was increased from group I to group IV. 'p' value 0.0251. The difference was statistically significant.

VLDL

The mean VLDL in the control group (I) was 27.6 with SD of 7.6. The mean VLDL was increased from group I to group IV. ‘p’ value 0.0251. The difference was statistically significant.

HDL

The mean HDL in the control group (I) was 54.2 with SD of 9.1. The mean HDL was decreased from group I to group IV. ‘p’ value 0.0208. The difference was statistically significant.

LDL

The mean LDL in the control group (I) was 110.4 with SD of 27.4. The mean LDL was increased from group I to group IV. ‘p’ value 0.0022. The difference was statistically significant.

Table 14 : Correlation of BMI and TC / HDL ratio

BMI Group	TC / HDL ratio	
	Mean	**SD**
I	3.73	1.24
II	4.47	1.23
III	4.65	1.21
IV	5.01	1.32
'p'	**0.0017** **Significant**	

The mean TC/HDL ratio in the control group (I) was 3.73 with SD of 1.24. The mean TC/HDL ratio is increased from group I to group IV. 'p' value 0.0017. The difference is statistically significant.

Table 15 : BMI and Dyslipidemia

BMI Group	Dyslipidemia			
	Present		Absent	
	No	%	No	%
I (20)	4	20	16	80
II (31)	10	32.3	21	67.7
III (28)	14	50	14	50
IV (21)	11	52.4	10	47.6
Mean BMI	29.13		26.38	
SD	4.48		3.3	
'p'	**0.0052** **Significant**			

Mean BMI in the dyslipidemic patients were 29.13 with SD of 4.48. and absents of dyslipidimia patients had mean BMI of 26.38 with SD of 3.3. 'p' value 0.0052. statistically significant dyslipidimia increasing from Group I to Group IV.

Table 16 : BMI and Fasting B.S

BMI Group	FBS	
	Mean	**SD**
I (20)	90.8	10.2
II (31)	96	11
III (28)	99.5	16.5
IV (21)	103.4	22.5
'p'	**0.049** **Significant**	

The mean Fasting Blood sugar in the control group (I) was 90.8 with SD of 10.2. The mean Fasting Blood sugar was increased from group I to group IV. 'p' value 0.049. The difference is statistically significant.

Table 17 : BMI and DM

BMI Group	DM			
	Present		Absent	
I (20)	1	5	19	95
II (31)	3	9.7	28	90.3
III (28)	4	14.3	24	85.7
IV (21)	5	23.8	16	76.2
Mean BMI	30.22		27.0	
SD	4.2		3.7	
'p'	**0.0063** **Significant**			

Mean BMI in the Diabetic group was 30.22 with SD 4.2 and for Non Diabetic group Mean BMI was 27.0 and SD 3.7. 'p' value is (0.0063) ,statistically significant. The prevalence of diabetes increases from Group I to Group IV (5% to 23.8%)

Table 18 : BMI and Smoking (among males)

BMI Group	Smoking			
	Present		Absent	
	Number	%	Number	%
I (9)	4	44.4	5	55.6
II (17)	3	17.6	14	82.4
III (13)	2	15.4	11	84.6
IV (10)	1	10	9	90
Mean BMI	25.2		28.1	
SD	3.9		3.8	
'p'	**0.0432** **Significant**			

Mean BMI among smokers were 25.2 with SD of 3.9 and for non smokers 28.1 with SD of 3.8. The 'p' value is (0.0432),statistically significant.

Table 19 : Relationship of Waist circumference with Dyslipidemia and Hypertension

Waist Circumference	Dyslipidemia		Hypertension	
	Present	Absent	Present	Absent
Mean	90.9	83.5	93.5	83.6
S.D.	6.2	5.9	5.4	5.5
'p'	0.0001 Significant		0.0001 Significant	

The mean waist circumference among dyslipidemic groups were 90.9 with SD of 6.2 and for non dyslipidemic groups were 83.5 with SD of 5.9. p' value 0.0001 statistically significant.

The mean waist circumference among Hypertensive groups were 93.5 with SD of 5.4 and for non hypertensive groups were 83.6 with SD of 5.5. p' value 0.0001. statistically significant.

Table 20 : Relationship of Waist / Hip ratio with Dyslipidemia and Hypertension

Waist / Hip ratio	**Dyslipidemia**		**Hypertension**	
	Present	**Absent**	**Present**	**Absent**
Mean	1	0.86	1.05	0.86
S.D.	0.14	0.06	0.14	0.05
'p'	**0.0001 Significant**		**0.0001 Significant**	

The mean waist hip ratio among dyslipidemic groups were 1.0 with SD of 0.14 and for non dyslipidemic groups were 0.86 with SD of 0.06. p' value 0.0001,statistically significant.

The waist circumference among hypertensive groups were 1.05 with SD of 0.14 and for non hypertensive groups were 0.86 with SD of 0.05.The p' value is (0.0001) statistically significant.

Table 21 : Lipid profile and HT

Lipid	Hypertension				'p'
	Present		Absent		
	Mean	SD	Mean	SD	
TGL	188.6	36.7	139.1	29.8	**0.0001 Significant**
VLDL	37.7	7.3	27.8	6.0	**0.0001 Significant**
HDL	41.1	8.4	50.8	9.2	**0.0001 Significant**
TC	235.3	28.4	202.7	20.9	**0.0001 Significant**
LDL	156.5	25.6	124.1	19.2	**0.0001 Significant**

In hypertensive patients TGL, TC and LDL is increased and HDL is decreased compared to non hypertensive patients.

Table 22 : Lipid profile and DM

Lipid	DM				'p'
	Present		Absent		
	Mean	SD	Mean	SD	
TGL	195.3	39.6	148.8	35.9	0.0029 Significant
VLDL	39.1	7.9	29.8	7.2	0.0029 Significant
HDL	40.1	6.7	48.8	9.9	0.008 Significant
TC	225.5	27.6	206.2	18.6	0.00396 Significant
LDL	142.3	29.3	133.0	25.6	0.3592 Not Significant

TC, TGL are significantly elevated in Diabetes patients compared to Non diabetic and HDL was decreased in Diabetic patients. Mean LDL Value was not significantly elevated in Diabetic patients.

DISCUSSION AND COMPARATIVE ANALYSIS

Obesity is a chronic and increasingly common health hazard. Traditionally, this was believed to be associated with affluent lifestyles in the West. However, obesity is a fast growing problem in developing countries and is now known to be associated with increased health risks. Several studies in India have shown that changes in dietary patterns, physical activity levels, life styles associated with affluence, and migration to urban areas are related to increasing risk of metabolic diseases such as type 2 diabetes mellitus, hypertension and dyslipidaemia .

The most widely used method to gauge obesity is the Body mass index (BMI) criteria are focus in obesity treatment recommendations,with different treatment cutoff points based up on the presence or absence of obesity-related comorbid disease .In addition, many patients with these metabolic diseases are either overweight or obese. Important relationship between BMI and metabolic metabolic disease is critical toward a better understanding of the underlying pathophysiological processes leading to excessive fat-related metabolic disease.

In this study 100 patients were included according to the inclusion and exclusion criteria. Among hundred patients, 80 Patients (40 Males

and 40 Females) whose BMI was ≥ 25 formed the Study Group who were divided into 3 Groups, according to BMI.

Group	BMI
II	≥ 25 – 26.9
III	≥27 – 29.9
IV	≥30

Control Group consisted of age matched 20 Patients (9 males and 11 Females) with BMI < 25 and designated as Group I.

Anthropometric results

The mean BMI in this Study group is 28.7. Control group mean BMI is 22.3.

According to Indian standards for BMI values (Classification of Overweight and obesity by Body mass index), BMI of our study group belongs to obesity class I (28.0-32.9)

The Nutrition Foundation of India (NFI) study showed that 32.3% of middle class males and 50% of middle class females in Delhi are obese.[4]

According to H. E. Bays et al (2007): The National Health And Nutrition Examination Surveys (NHANES) showed mean BMI was 27.9 compared to 28.9 in this study and " The Study To Help Improve Early Evaluation and Management of Risk Factors Leading to Diabetes (SHIELD) Survey" mean BMI was 27.8 .

In both studies Prevalence of diabetes mellitus, hypertension, and dyslipidemia occurred among all obese subjects, but the prevalence correlated with increase in BMI[54].

Study by H Shukla et al (2002) – In this study (cross- sectional) representative surveys were carried out in the city of Mumbai in Western India. Results showed 19% of men and women were thin built (BMI<18.5 kg/m^2), while 19% of men and 30% of women were overweight (BMI≥25kg/m^2)[55].

Pragti Chhabra, Sunil K. Chhabra et al - In their study, overall only half (49.7%) of the subjects had a normal nutritional status, while 24.8% were underweight, 19.4% overweight, and 6.1%. obese[56]**.**

Ron T Varghese, Vijayakumar K et al - In their study of pattern of obesity across different age groups in a rural setting showed that 30-40 % of the population was either obese or overweight[57]**.**

BMI and Smoking (among males) .

In our study mean BMI among smokers were lower (25.2) than the non smokers (28.1) . According to **Mangesh S ,Pednekar et al(2006)** in their study "Association between tobacco use and body mass index in urban Indian population: implications for public health in India" , showed that all forms of tobacco use were associated with low BMI. The prevalence of low BMI was highest in bidi-smokers (32% compared to 13% in non-users) [58]

HYPERTENSION IN OBESITY

In this study parameters like hypertension (Systolic& Diastolic B.P), was analysed among all BMI groups.

BMI and systolic B.P

Mean systolic Blood pressure on comparison showed 115.0 mm Hg in group I while in group II, III and IV it was 125, 129.3 and 134.9 respectively. Mean Systolic BP increased from Group I to Group IV.

BMI and Diastolic B.P

Mean Diastolic Blood pressure was compared among the different groups Mean Diastolic BP in BMI group I (control group) is 71.9 while

in group II, III and IV it was 76.3 , 79.1 and 84.6 respectively. Mean Diastolic BP increased from Group I to Group IV.

Brown et al study - four categories of BMI (<25, 25≤27, 27 ≤30, and ≥30) were compared for hypertension and Dyslipidemia. The results showed that mean systolic and diastolic blood pressures increased with increasing BMI in men and women. Systolic blood pressure increased from 121.3 to 130.7 mm Hg in males and 115.7 to 127.3 mm Hg in women according to low BMI to high BMI group. Similarly, Diastolic BP also increased from 74.0 to 80.9 mm Hg in males and 69.7 to 76.0mmHg in women according to low BMI to high BMI group[32]. Our study results are similar to this study.

Among men the prevalence of high blood pressure increased progressively with increasing BMI from 15% at a BMI of <25 to 42% with BMI of ≥30 .Prevalence of high blood pressure increased progressively from 15% among women with BMI of <25 to 38% among women with BMI of >30.[33]

In NHANES III, the age-adjusted prevalence of hypertension (defined as systolic blood pressure >140 mm Hg, diastolic blood pressure >90 mm Hg, or use of antihypertensive medication) in obese men and women was 42% and 38%, respectively. These prevalence rates are more than twice of the prevalence of hypertension in lean men and women

(15% prevalence rate in both men and women)[59, 60] . The risk of hypertension also increases with weight gain .

Among subjects followed in the **Framingham Study**, there was a.6.5-mm Hg increase in blood pressure with every 10% increase in body weight. [33]

Weight gain, even to levels not considered to be a problem, increases the incidence of hypertension. This was clearly shown in a **Nurses' Health Study (Huang et al., 1998**) .Those women, now in midlife, who had gained as little as 5 kg over their weight at age 18 had a 60% higher relative risk of developing hypertension than did those whose weight had not changed more than 2 kg.[16]

According to H. E. Bays et al SHIELD study showed that 80% of hypertensive patients were overweight or obese[54].

Diabetes correlation with BMI

Mean BMI in the Diabetic subjects were 30.22 compared to the non Diabetic whose Mean BMI was 27. The 'p' value (0.0063 was statistically significant. The prevalence of diabetes on comparison showed 5 % in group I while in group II, III and IV it was 9.7 , 14.3 and 23.8 respectively .

The prevalence of diabetes increases from lowest BMI Group I to Highest BMI Group IV (5% to 23.8%).

Harris et al study showed that (NHANES III), two thirds of the men and women in the United States with diagnosed type 2 diabetes have a BMI of 27.0 kg/m^2 or greater.[27]

Flegal KM et al in their study, the risk of diabetes increases linearly with BMI: the prevalence of diabetes increased from 2% in those with a BMI of 25.0 to 29.9 kg/m^2, to 8% in those with a BMI of 30 to 34.9 kg/m^2, and to 13% in those with a BMI greater than 35 kg/m^2.[2, 3]

In the "**Nurses' Health Study**", the risk of diabetes began to increase when BMI exceeded the "normal" value of 22 kg/m^2 .

Ohlson L.O et al, Lundgren. H et al and . Kaye et al in their studies showed that the risk of diabetes also increases with weight gain during adulthood. Among men and women aged 35 to 60 years, the risk of diabetes was three times greater in those who gained 5 to 10 kg since the age of 18 to 20 years than in those who maintained their weight within 2 kg.[28,29,30.]

Lipid profile in correlation with BMI

TC

In this study the mean total cholesterol in the control group (I) was 192.2 . The mean total cholesterol was increased from group I to group IV .

Brown et al . reported that mean serum cholesterol level increased with increasing BMI from 193mg/dl among men at the lowest BMI category to 211mg/dl in men at the highest BMI category .Among women TC levels increased from 195mg/dl to 217mg/dl.[32]

In our study the TC in males among group I was 189.2 and 209.3 in(highest BMI) group IV. Mean total cholesterol increased from Group I to Group IV in males.

In women TC in group I was 194.7 and 218.6 in group III and 218.3 in group IV . In females Mean total cholesterol increased from Group I to Group III and plateaued at Group I V .

This is comparable to Brown et al that, in men, there was a progressive increase in the prevalence of hypercholesterolemia (total blood cholesterol >240 mg dL or 6.21 mmol/L) with increasing BMI. In women, by contrast, the prevalence of hypercholesterolemia was highest

at a BMI of 25.0 to 27.0 kg/m^2, and it did not increase further at higher BMI values.

Hanumanthappa Nandeesha, et al in their study total cholesterol, triglyceride and LDL cholesterol were found to be significantly increased in overweight and obese subjects and HDL cholesterol was significantly decreased in obese subjects[59].

HDL

The mean HDL in the control group (I) was 54.2 with SD of 9.1. The mean HDL decreased from group I to group IV.

Brown et al in their study showed that mean HDL WITH BMI <25 WAS 50mg/dl and it declined to 40mg/dl at a BMI of ≥30 .Among women the age adjusted mean HDL level decreased from 50mg/dl with BMI of <25 to 49mg/dl for women with BMI of ≥30.

In our study also showed that mean HDL in group I (BMI <25) was 54.2 with SD 9.1 in group IV(≥30) mean HDL was 44.7 with SD of 10.1 HDL progressively decreased from group I to group IV.

Dongsheng Hu et al in their Strong Heart Study there was a 60% increase in BMI (from 25 to 40 kg/m^2) was associated with a 6-mg/dL

decrease in HDL cholesterol in women,and 12-mg/dL decrease in HDL cholesterol in men .[60]

The main lipoprotein abnormality related to obesity in American Indians was decreased HDL cholesterol, especially in men. Central adiposity was more associated with abnormal lipid/lipoprotein profiles than general obesity in women; both were equally important in men.

Bhatti MS et al 2001 , in their study regarding lipid profile in obesity showed that obese persons total lipids, total cholesterol, , LDL, VLDL, chylomicrons LDL/HDL, VLDL/HDL, TG/HDL and TC/HDL ratios level showed significant increase while HDL level was significantly decreased.[61]

TGL

The mean TGL (triglyceride) in the control group (I) was 138.2 with SD of 38. The mean TGL was increased from group I to group IV.

Domenico Sommariva et al in their study showed that body mass index is positively correlated with serum triglycerides, VLDL lipids, HDL-triglycerides are* negatively correlated with HDL-cholesterol.[62]

In the **Strong Heart Study population , by Dongsheng Hu et al** showed that 60% increase in BMI (from 25 to 40 kg/m^2) was associated

with a 5-mg/dL increase in triglyceride in women and but a 74-mg/dL increase in triglycerides in men.[60]

Bhatti MS et al in their study regarding lipid profile in obesity noticed that Direct correlation between plasma triglycerides and body weight [61.]

LDL

The mean LDL in the control group (I) was 110.4 with SD of 27.4. The mean LDL was increased from group I to group IV.

Misra A. et al : "Prevalence of diabetes, obesity and dyslipidaemia in urban slum population in northern India" among 532 subjects (170 males and 362 females) showed that total cholesterol and low-density lipoprotein cholesterol were high in both males and females.[63]

Ronald M. Krauss, et al ,in their study "Obesity -Impact on Cardiovascular Disease" showed that obesity has a strong effect on lipoprotein metabolism, regardless of ethnic group. Increased weight is a determinant of higher levels of triglycerides, elevated LDL-C, and low HDL-C.. The association between obesity and LDL-C is more complex. LDL-C concentrations increase with BMI in men, but such increases are not as pronounced in women, the elderly, and some ethnic groups. Increasing BMI is associated with small, atherogenic LDL. Furthermore,

central obesity in women is associated with elevated LDL-C concentrations.[64]

TC/HDL ratio.

The mean TC/HDL ratio in the control group (I) was 3.73 with SD of 1.24. In Group IV it was 5.01 with SD of 1.32. The mean TC/HDL ratio was increased from group I to group IV. 'p' value 0.0017. The difference was statistically significant.

Jean-Pierre et al in their study " Evaluation and management of atherogenic dyslipidemia: beyond low-density lipoprotein cholesterol" Study shows that elevation of the total cholesterol to HDL-C ratio in the absence of marked elevation of LDL-C level is a salient feature of insulin-resistance , among abdominal obesity patients, as well as of type 2 diabetic patients.Among men there was a high prevalence of the metabolic syndrome (40%).

BMI and Dyslipidemia

Mean BMI in the dyslipidemic patients were s 26.38 with SD of 3.3.The 'p' value(0.0052.)is statistically significant. Dyslipidemia showed increasing pattern from Group I to Group IV. (20 % to 52.4 %) .
In NHANES STUDY 84% of dyslipidaemic patients were overweight or obese[54].In SHIELD STUDY 75% of dyslipidaemic patients were overweight or obese SHIELD and NHANES demonstrated that a

gradual increases in dyslipidaemia is seen with progression of BMI till 30 kg/m^2 . Beyond BMI of 30 kg/m^2 prevalence plataued and in fact estimates began to decline in NHANES. [54]

Waist circumference correlation with Dyslipidemia and Hypertension

The mean waist circumference among dyslipidemic groups were 90.9 and for non dyslipidemic groups were 83.5 .The p' value(0.0001) is statistically significant in our study.

The mean waist circumference among hypertensive groups were 93.5 and 83.6 for non hypertensive groups .The p' value (0.0001) statistically significant in our study.

Okosun et al,1998 study showed that Waist circumference was positively correlated with blood pressure and fasting blood glucose. Substantial reduction in hypertension and diabetes in men and women is achievable if the waist size is decreased in these populations.[67]

Ian Janssen et al 2004.,study showed that the addition of waist circumference (WC) to body mass index (BMI; in kg/m^2) predicts a greater variance in health risk than does BMI alone.[67]

Waist Hip ratio with Dyslipidemia and Hypertension

The mean waist hip ratio among dyslipidemic groups were 1.0 and for non dyslipidemic groups were 0.86 .The p' value is statistically significant.

The waist circumference among hypertensive groups were 1.05 and for non hypertensive groups were 0.86 . The p' value is statistically significant**.**

Yusuf S et al study, 2005 showed that waist-to-hip ratio of more than 0.83 for women and 0.9 for men would result in a 3-fold increase in population attributable risk for myocardial infarction. This is particularly important in regions such as Asia, which have not had significant problems with obesity as measured by BMI but would have considerably greater cardiovascular risk if waist-to-hip ratio was used.[68]

Jern et al, 1992 study .The aims were to investigate the influence of obesity (as defined by body mass index) and abdominal fat accumulation (as defined by the waist/hip ratio) on hemodynamics at rest and during mental stress. Cardiac output and stroke volume were positively correlated to body mass index, but inversely to waist/hip ratio).Total

peripheral resistance during stress correlated inversely to body mass index whereas high waist/hip ratio was associated with higher systemic vascular resistance .[69]

Lipid profile abnormalities in Hypertension

In this study TC ,TGL, LDL and TC:HDL ratio were increased and HDL level were decreased among the hypertensive subjects* compared to the non hypertensive subjects.

Our study was supported by the **Thakur AK et al ' Study of lipid level in uncomplicated Hypertension**'. In their study mean total cholesterol and TC:HDL ratio were higher among hypertensives.[70]

Ruben O. Halperin et al in their study " **Dyslipidemia** and the Risk of Incident Hypertension in Men demonstrated higher levels of plasma TC, non-HDL-C, and the TC/HDL-C ratio are independently associated with a subsequent increased risk of incident hypertension in apparently healthy men and that higher levels of HDL-C are associated with a decreased risk of incident hypertension**.**[71]

Lipid profile in Diabetic patients.

TC, TGL are significantly elevated in Diabetes patients compared to non diabetic and HDL was decreased in Diabetic patients. Mean LDL Value was not significantly elevated in Diabetic patients.

H.B. Chandalia et al in their study " LIPID ABNORMALITIES IN DIABETES MELLITUS" reported that Plasma cholesterol and triglycerides were significantly elevated in diabetics compared to non-diabetics. Reduction of HDL cholesterol (by 10-20%), primarily due to a fall of HDL cholesterol is also seen.

The most frequent serum lipid abnormality in Type2 diabetes is an elevation of serum triglycerides to 1.5 - 3.0 times non-diabetic controls.[72]

Jim Nuovo et al demonstrated hyperlipidemia in patients with Type 2 Diabetes in their study . The most common lipid abnormalities in these patients include hypertriglyceridemia and reduced high-density lipoprotein (HDL) cholesterol levels.[73]

CONCLUSION

- The prevalence of high blood pressure and mean levels of systolic and diastolic blood pressure are increased among increased BMI groups.

- As BMI increases total cholesterol, LDL,TGL, TC/HDL ratio increases and HDL decreases.

- Dyslipidemia is more common among obese patients compared to patients with normal body mass index.

- In general, the presence of hypertension, high total cholesterol, LDL, TGL, TC/HDL ratio and low HDL shows increasing trend with increasing BMI, as does the combined prevalence of both hypertension and dyslipidemia
- The prevalence of diabetes is increased as the BMI increases

SUMMARY

The study **"DYSLIPIDEMIA AND HYPERTENSION IN OBESE PATIENTS WITH CORRELATION TO BODY MASS INDEX"** is a cross- sectional study among patients attending medical Outpatient Department and Medical Wards in Govt Rajaji Hospital for varying illness , 80 Patients (40 Males and 40 Females) whose BMI were ≥ 25 selected for the study Group(Group II ,III ,IV) and 20 Patients (9 males and 11 Females) whose BMI < 25 were selected in the same age group as controls (Group I).

Subjects divided into four groups according to BMI

Group	BMI	
I	□25	**Healthy weight**
II	≥ 25 – 26.9	**Overweight**
III	≥27 – 29.9	**Overweight**
IV	≥30	**Obesity**

Patients and controls who satisfied the inclusion and exclusion criteria underwent various investigations like fasting blood sugar, and lipid profile were taken. Relationship of various anthropometric measurements like body mass index ,waist circumference, waist hip ratio were

analyzed. A high BMI showed significant correlation with hypertension, diabetes and dyslipidemia.

Thus our study highlights the significance of a simple measurement of BMI in day to day clinical practice in detecting the patients with high cardiovascular risk like hypertension, diabetes, obesity, dyslipidemia and metabolic syndrome. The cut off values for and BMI, waist circumference, and waist hip ratio should be defined according to the different ethnic populations. This will help in early detection of the people at risk so that we can advice healthy life style and nutritional habits for the high risk group and start specific therapy when ever necessary.

APPENDIX-1

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APPENDIX-2

GLOSSARY

BMI	- Body Mass Index
BP	- Blood Pressure
CAD	- Coronary Artery Disease
CHD	- Coronary Heart Disease
CM	- Chylomicrone
CRP	- C Reactive Protein
CVD	- Cardio Vascular Disease
DM	- Diabetes Mellitus
DBP	- Diastolic Blood Pressure
ECG	- Electro Cardiogram
EF	- Ejection Fraction
F/H	- Family History
HT	- Hypertension
HDL	- High Density Lipoprotein
HC	- Hip Circumference
IR	- Insulin Resistance
LDL	- Low Density Lipoprotein
Lp (a)	- Lipoprotein (a)
MS	- Metabolic Syndrome

NCEP- ATP III	- National Cholesterol Education Programme. Adult Treatment Panel – III
IDF	- International Diabetic Federation
OCP	- Oral Contraceptive Pill
SBP	- Systolic Blood Pressure
TC	- Total Cholesterol
TG	- Triglyceride
VLDL	- Very Low Density Lipoprotein
WC	- Waist Circumference
WHO	- World Health Organisation
WHR	- Waist Hip Ratio

APPENDEX-3

PROFORMA

DYSLIPIDEMIA AND HYPERTENSION IN OBESE PATIENTS

WITH CORRELATION TO BODY MASS INDEX

1. **Sl. No :**
2. **Name :**
3. **IP/OP No :**
4. **Age :**
5. **Domicile : Urban/Rural**
6. **Life Style : Sedentary / Non Sedentary**
7. **Diet : Vegetarian / Non Veg.**
8. **Smoking : present /Absent.**
9. **Past History : HT / DM / CVA / CAD / Others**
10. **Drug History : OCP/ Steroids / Beta blocker/ Frusemide / Others**
11. **Examination : Anthropometry**

Height (in cm)	**Weight (in kgs)**	**BMI Kg/m^2**	**Waist (in cm) Circumference**	**Hip (in cm) Circumference**	**W /H Ratio**

G.E : Pallar / Edema / Cyanosis / Jaundice / Clubbing

CVS : PR : BP : JVP :

S1 S2 S3 S4

RS :

Abdomen :

17. Investigation :

FBS

Fasting lipid profile

Total Cholesterol	T.G	HDL	VLDL	LDL

APPENDIX-4
MASTER CHART

S.No	Age	Sex	Group	Smoking	Hight (in cm)	Weight	BMI	WC	HC	W/H Ratio	HT	SBP	DBP	DM	FBS	TGL	VLDL	HDL	TC	LDL	TC:HDLC Ratio
1	29	Male	Control	1	163	55	20.7	75.0	86.2	0.87	2	102	70	2	89	131	26	60	196	110	3.27
2	29	Female	Control	2	148	50	22.8	73.0	92.4	0.79	2	100	60	2	86	122	24	65	195	106	3.01
3	41	Female	Control	2	145	43	20.5	77.0	100.0	0.77	2	114	64	2	82	116	23	65	185	97	2.85
4	38	male	Control	1	156	53	21.8	81.0	89.0	0.91	2	118	64	2	85	121	24	50	188	114	3.76
5	52	Male	Control	1	162	51	19.4	84.0	98.8	0.85	2	112	72	2	87	121	24	47	181	110	3.86
6	42	Female	Control	2	148	54	24.7	88.0	91.7	0.96	2	114	66	2	130	220	44	38	224	142	5.89
7	34	Female	Control	2	146	42	19.7	74.0	91.4	0.81	2	100	64	2	86	115	23	60	167	84	2.78
8	37	Female	Control	2	156	52	21.4	75.0	98.7	0.76	2	102	60	2	88	120	24	66	184	94	2.79
9	72	Male	Control	2	161	58	22.4	82.0	98.8	0.83	2	130	80	2	92	115	23	51	162	88	3.18
10	41	Female	Control	2	152	57	24.7	71.0	87.7	0.81	2	100	68	2	90	119	24	60	166	82	2.76
11	45	Female	Control	2	154	53	22.3	87.0	95.6	0.91	1	150	96	2	96	189	38	48	256	170	5.33
12	56	Male	Control	1	160	62	24.2	76.0	85.4	0.89	2	124	84	2	88	121	24	50	183	109	3.66
13	43	Male	Control	2	158	59	23.6	85.0	96.6	0.88	2	120	76	2	94	115	23	53	181	105	3.42
14	51	Male	Control	2	158	60	24.0	92.0	83.6	1.10	1	150	96	2	99	210	42	38	248	168	6.53
15	27	Female	Control	2	151	47	20.6	76.0	92.7	0.82	2	98	60	2	84	117	23	58	170	89	2.94
16	53	Female	Control	2	150	52	23.1	79.0	108.2	0.73	2	108	66	2	86	118	24	63	170	83	2.70
17	53	Female	Control	2	155	54	22.5	88.0	94.6	0.93	2	140	92	2	91	224	45	38	241	158	6.34
18	33	Male	Control	2	162	63	24.0	87.0	107.4	0.81	2	100	70	2	87	129	26	58	190	106	3.27
19	26	Male	Control	2	152	49	21.2	78.0	91.8	0.85	2	108	60	2	83	119	24	52	173	97	3.33
20	33	Female	Control	2	148	51	23.3	80.0	102.6	0.78	2	106	70	2	93	121	24	63	183	96	2.91
21	50	Female	T	2	152	82	35.5	97.0	81.5	1.19	2	158	102	2	117	225	45	35	197	117	5.63
22	29	Female	T	2	157	79	32.0	86.0	96.6	0.89	2	114	70	2	88	125	25	50	222	147	4.44
23	55	Male	T	2	153	63	26.9	84.0	89.4	0.94	2	122	64	2	81	170	34	46	180	100	3.91

24	66	Female	T	2	151	62	27.2	90.0	107.1	0.84	2	128	72	2	98	126	25	66	207	116	3.14
25	39	Male	T	2	160	70	27.3	91.0	100.0	0.91	2	122	72	2	118	124	25	50	186	111	3.72
26	38	Female	T	2	148	60	27.4	82.0	94.3	0.87	2	112	64	2	130	240	48	39	203	116	5.21
27	29	Male	T	1	158	64	25.6	84.0	100.0	0.84	2	110	72	2	94	109	22	50	196	124	3.92
28	22	Male	T	2	157	64	26.0	83.0	96.5	0.86	2	122	60	2	98	136	27	44	200	129	4.55
29	38	Male	T	2	150	67	29.8	82.0	92.1	0.89	2	116	66	2	128	235	47	36	201	118	5.58
30	45	Male	T	1	147	60	27.8	95.0	95.0	1.00	1	166	98	2	89	135	27	50	242	165	4.84
31	38	Male	T	2	149	61	27.5	86.0	98.9	0.87	2	110	64	2	95	130	26	58	212	128	3.66
32	43	Male	T	2	149	64	28.8	90.0	96.8	0.93	2	130	86	2	88	131	26	45	190	119	4.23
33	28	Female	T	2	149	72	32.4	92.0	100.0	0.92	2	102	64	2	86	150	30	34	229	165	6.74
34	73	Male	T	2	150	75	33.3	89.0	86.4	1.03	2	128	88	2	93	135	27	43	180	110	4.19
35	46	Male	T	2	160	71	27.7	86.0	95.6	0.90	2	112	74	2	90	141	28	53	221	140	4.17
36	25	Male	T	2	156	64	26.3	85.0	109.0	0.78	2	112	72	2	102	120	24	50	170	96	3.40
37	47	Female	T	2	163	74	27.9	85.0	97.7	0.87	2	116	62	2	93	115	23	65	205	117	3.15
38	33	Female	T	2	141	58	29.2	86.0	98.9	0.87	2	108	64	2	94	140	28	52	242	162	4.65
39	44	Female	T	2	157	75	30.4	88.0	95.7	0.92	2	116	66	2	89	120	24	66	205	115	3.11
40	37	Female	T	2	145	84	40.0	97.0	93.3	1.04	2	192	110	1	140	210	42	37	250	171	6.76
41	63	Male	T	2	159	67	26.5	80.0	87.9	0.91	2	122	74	2	105	130	26	60	172	86	2.87
42	46	Female	T	2	145	53	25.2	87.0	107.4	0.81	2	116	64	2	83	120	24	66	183	93	2.77
43	38	Female	T	2	151	58	25.4	88.0	101.1	0.87	2	114	60	2	89	111	22	60	216	134	3.60
44	51	Male	T	2	151	60	26.3	88.0	94.6	0.93	2	118	78	2	97	121	24	49	175	102	3.58
45	42	Male	T	2	150	60	26.7	93.0	95.9	0.97	1	154	92	1	128	210	42	38	250	170	6.58
46	44	Female	T	2	147	61	28.2	92.0	98.9	0.93	2	146	98	1	139	205	41	36	183	106	5.08
47	43	Female	T	2	140	72	36.7	99.0	92.5	1.07	1	176	106	2	86	215	43	42	265	180	6.31
48	50	Male	T	2	144	65	31.3	91.0	97.8	0.93	2	112	68	2	95	125	25	60	210	125	3.50
49	47	Female	T	2	146	60	28.1	91.0	100.0	0.91	2	152	102	2	90	130	26	38	181	117	4.76
50	60	Male	T	2	162	70	26.7	84.0	98.8	0.85	2	124	68	2	89	146	29	48	212	135	4.42
51	61	Male	T	1	156	61	25.1	95.0	73.1	1.30	2	148	98	2	111	115	23	37	232	172	6.27
52	35	Female	T	2	152	63	27.3	84.0	98.8	0.85	2	118	66	2	92	115	23	60	208	125	3.47
53	67	Female	T	2	143	53	25.9	89.0	90.8	0.98	2	158	100	2	104	153	31	38	241	172	6.33
54	65	Female	T	2	148	60	27.4	81.0	97.6	0.83	2	106	66	2	90	120	24	60	234	150	3.90

55	27	Female	T	2	159	76	30.1	96.0	105.5	0.91	2	104	74	2	80	130	26	60	209	123	3.48
56	34	Male	T	1	152	75	32.5	102.0	72.9	1.40	2	146	96	2	90	210	42	32	194	120	6.06
57	49	Male	T	2	159	102	40.3	96.0	100.0	0.96	2	196	104	2	85	220	44	40	256	172	6.40
58	39	Male	T	2	155	68	28.3	81.0	97.6	0.83	2	118	82	2	90	149	30	42	178	106	4.23
59	36	Male	T	2	164	77	28.6	84.0	86.6	0.97	2	122	70	2	92	120	24	48	179	107	3.73
60	55	Male	T	2	150	69	30.7	96.0	105.5	0.91	2	132	84	2	94	125	25	46	173	102	3.76
61	33	Female	T	2	145	68	32.3	98.0	99.0	0.99	2	150	100	2	89	166	33	58	223	132	3.84
62	33	Female	T	2	145	76	36.1	91.0	104.6	0.87	2	106	76	2	145	210	42	58	250	150	4.31
63	56	Female	T	2	144	60	28.9	79.0	96.3	0.82	2	114	66	2	91	125	25	38	210	147	5.53
64	67	Male	T	2	153	68	29.0	81.0	85.3	0.95	2	134	62	2	89	214	43	34	204	127	5.99
65	29	Female	T	2	152	69	29.9	83.0	100.0	0.83	2	102	62	2	97	121	24	55	216	137	3.93
66	55	Male	T	2	158	92	36.9	100.0	95.2	1.05	1	170	110	1	135	161	32	42	244	170	5.81
67	43	Male	T	2	153	74	31.6	91.0	94.8	0.96	2	116	72	2	88	144	29	42	184	113	4.38
68	51	Male	T	2	162	90	34.3	103.0	86.6	1.19	2	158	100	2	110	215	43	32	257	182	8.03
69	71	Male	T	2	159	65	25.7	96.0	87.3	1.10	1	164	102	2	104	210	42	50	256	164	5.12
70	37	Male	T	2	150	58	25.8	87.0	107.4	0.81	2	122	70	2	84	135	27	45	181	109	4.02
71	25	Female	T	2	159	67	26.5	78.0	94.0	0.83	2	102	62	2	82	145	29	54	241	158	4.46
72	37	Female	T	2	155	64	26.6	84.0	98.8	0.85	2	120	62	2	130	205	41	37	232	154	6.27
73	34	Female	T	2	148	59	26.9	74.0	89.2	0.83	2	110	72	2	87	110	22	62	174	90	2.81
74	55	Male	T	2	150	61	27.1	98.0	86.0	1.14	2	174	106	2	105	210	42	30	236	164	7.87
75	51	Female	T	2	144	57	27.5	80.0	93.0	0.86	2	122	68	2	106	145	29	58	181	94	3.12
76	72	Female	T	2	146	60	28.1	93.0	82.3	1.13	1	164	108	2	91	210	42	60	268	166	4.47
77	29	Male	T	2	154	67	28.3	85.0	100.0	0.85	2	118	68	2	93	140	28	41	194	125	4.73
78	63	Male	T	2	157	78	31.6	90.0	98.9	0.91	2	116	70	2	88	145	29	41	188	118	4.59
79	36	Male	T	2	150	73	32.4	87.0	100.0	0.87	2	108	72	2	146	151	30	42	207	135	4.93
80	62	Male	T	2	163	70	26.3	79.0	89.8	0.88	2	122	68	2	86	145	29	45	206	132	4.58
81	55	Male	T	2	156	65	26.7	76.0	85.4	0.89	2	112	84	2	99	129	26	50	208	132	4.16
82	50	Female	T	2	152	62	26.8	76.0	96.2	0.79	2	116	64	2	85	130	26	60	201	115	3.35
83	65	Female	T	2	150	61	27.1	92.0	93.9	0.98	1	164	104	2	100	210	42	36	262	184	7.28
84	29	Male	T	2	154	65	27.4	91.0	107.1	0.85	2	102	88	2	83	165	33	48	206	125	4.29
85	61	Female	T	2	152	64	27.7	86.0	97.7	0.88	2	110	64	2	86	160	32	60	244	152	4.07

86	68	Female	T	2	142	52	25.8	83.0	102.5	0.81	2	124	82	2	94	120	24	62	187	101	3.02
87	21	Male	T	2	156	61	25.1	89.0	102.3	0.87	2	110	64	2	79	130	26	48	200	126	4.17
88	45	Female	T	2	134	46	25.6	87.0	91.6	0.95	1	160	102	2	134	220	44	52	262	166	5.04
89	53	Male	T	1	161	73	28.2	97.0	72.4	1.34	1	162	114	1	143	215	43	38	261	180	6.87
90	65	Female	T	2	153	73	31.2	87.0	102.4	0.85	2	112	64	2	94	115	23	44	186	119	4.23
91	27	Female	T	2	142	52	25.8	80.0	95.2	0.84	2	100	58	2	85	121	24	62	211	125	3.41
92	42	Male	T	2	151	61	26.8	82.0	91.1	0.90	2	110	70	2	86	135	27	48	218	143	4.54
93	43	Female	T	2	148	60	27.4	94.0	93.1	1.01	2	172	98	2	86	120	24	55	249	170	4.53
94	38	Female	T	2	142	61	30.3	90.0	98.9	0.91	2	120	80	2	133	116	23	35	165	107	4.72
95	40	Female	T	2	152	60	26.0	88.0	80.0	1.10	2	142	98	2	102	200	40	35	245	170	7.00
96	25	Female	T	2	150	60	26.7	83.0	101.2	0.82	2	106	60	2	89	130	26	55	231	150	4.20
97	33	Female	T	2	141	53	26.7	86.0	100.0	0.86	2	146	94	2	92	210	42	36	182	104	5.06
98	51	Male	T	1	164	71	26.4	93.0	96.9	0.96	1	158	108	2	100	210	42	35	245	168	7.00
99	31	Female	T	2	156	64	26.3	79.0	100.0	0.79	2	110	60	2	82	109	22	62	223	139	3.59
100	42	Male	T	2	170	76	26.3	79.0	101.3	0.78	2	122	82	2	95	130	26	45	201	130	4.47

KEYS TO MASTER CHART

Smoking

1- smoker

2- non smoker

BMI- Body mass index

WC – Waist Circumference in cms

HC- Hip Circumference

W/H-Waist Hip ratio

HT- Hypertension

1- Present

2- Absent

SBP- Systolic Blood Pressure

DBP-Diastolic Blood Pressure

DM- Diabetes Mellitus

1- Present

2- Absent

FBS- Fasting Blood Sugar

HDL- High Density Lipoprotein

TGL - Triglycerides

VLDL – Very Low Density Lipoprotein

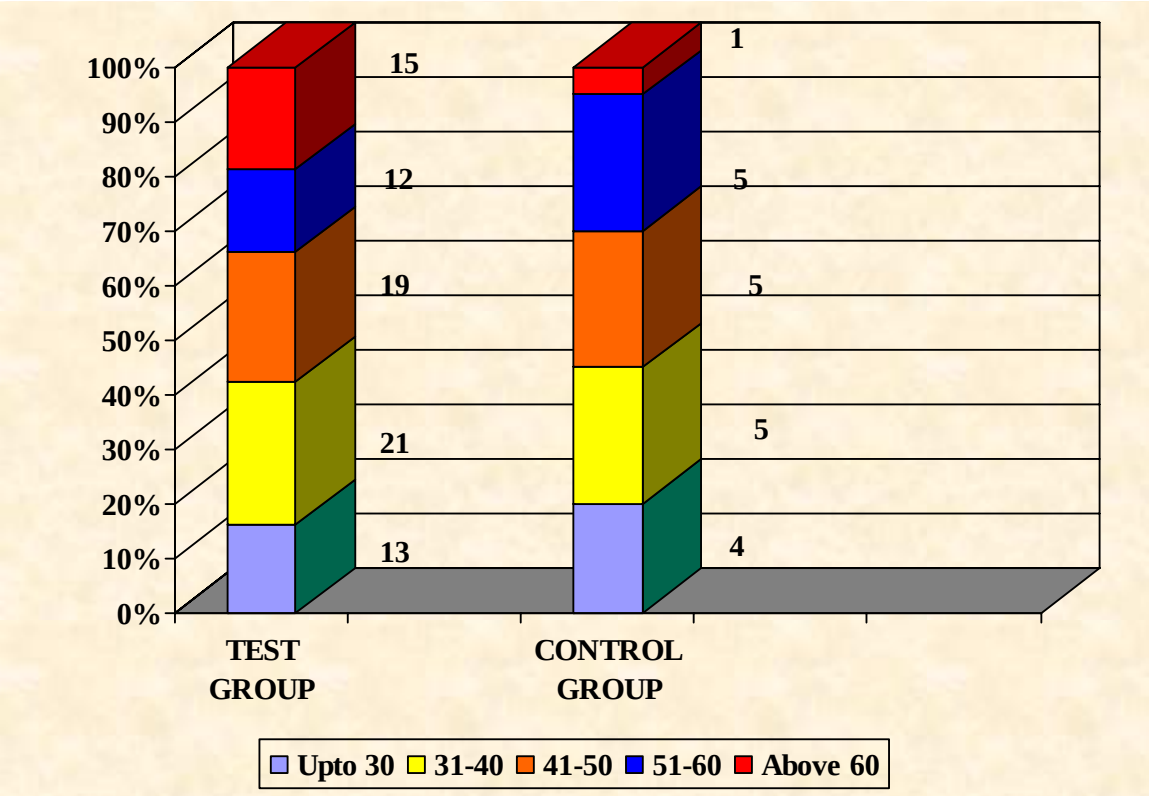
LDL - Low Density Lipoprotein

TC- Total Cholesterol

TC/HDL – Total Cholesterol/ High Density Lipoprotein

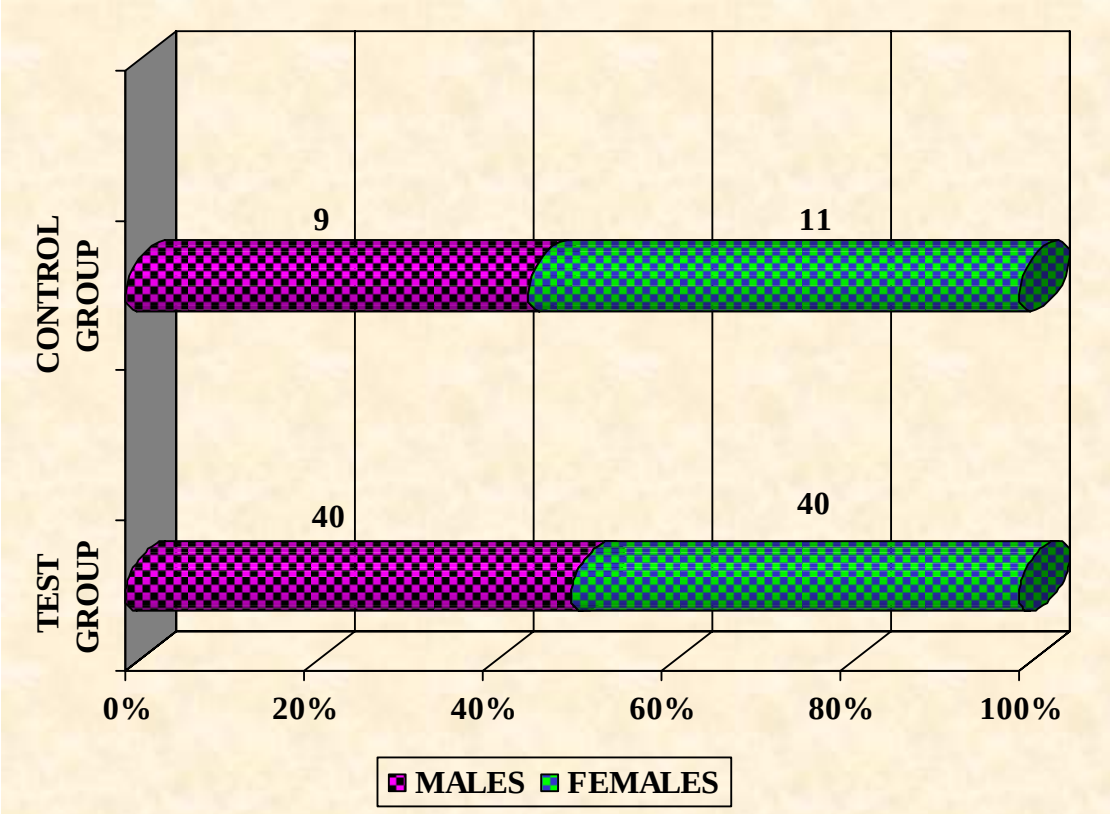
AGE DISTRIBUTION

FIG:2



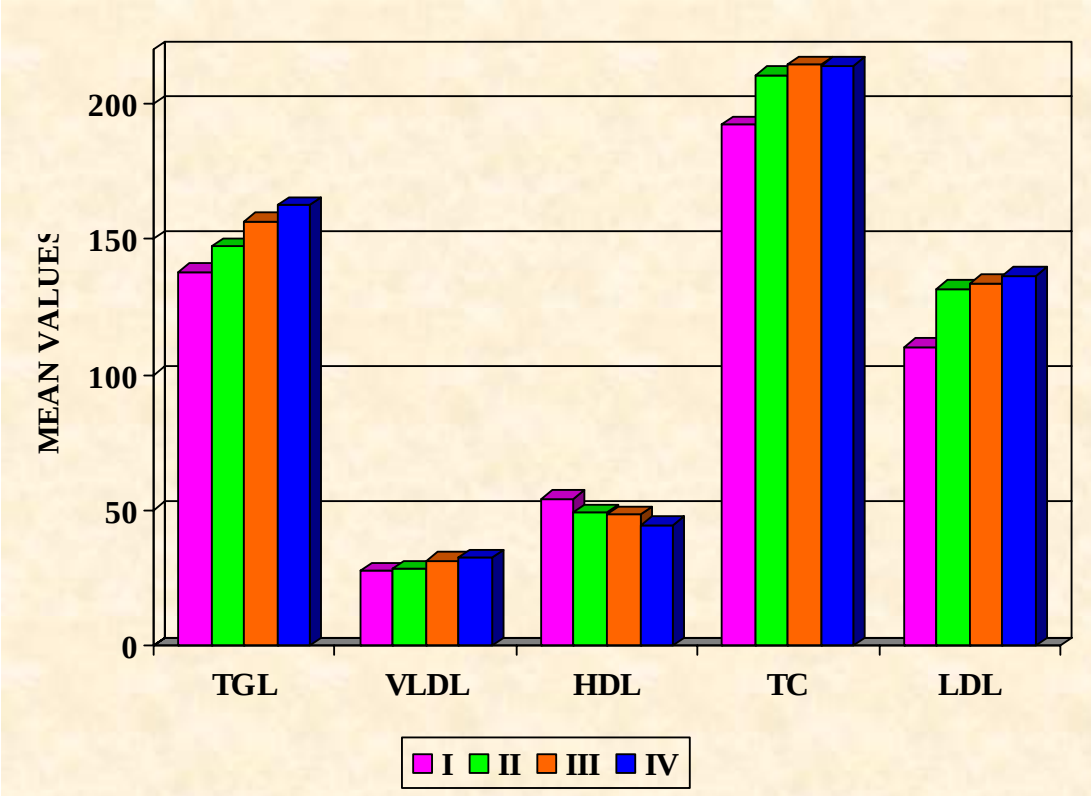
SEX DISTRIBUTION

FIG:3



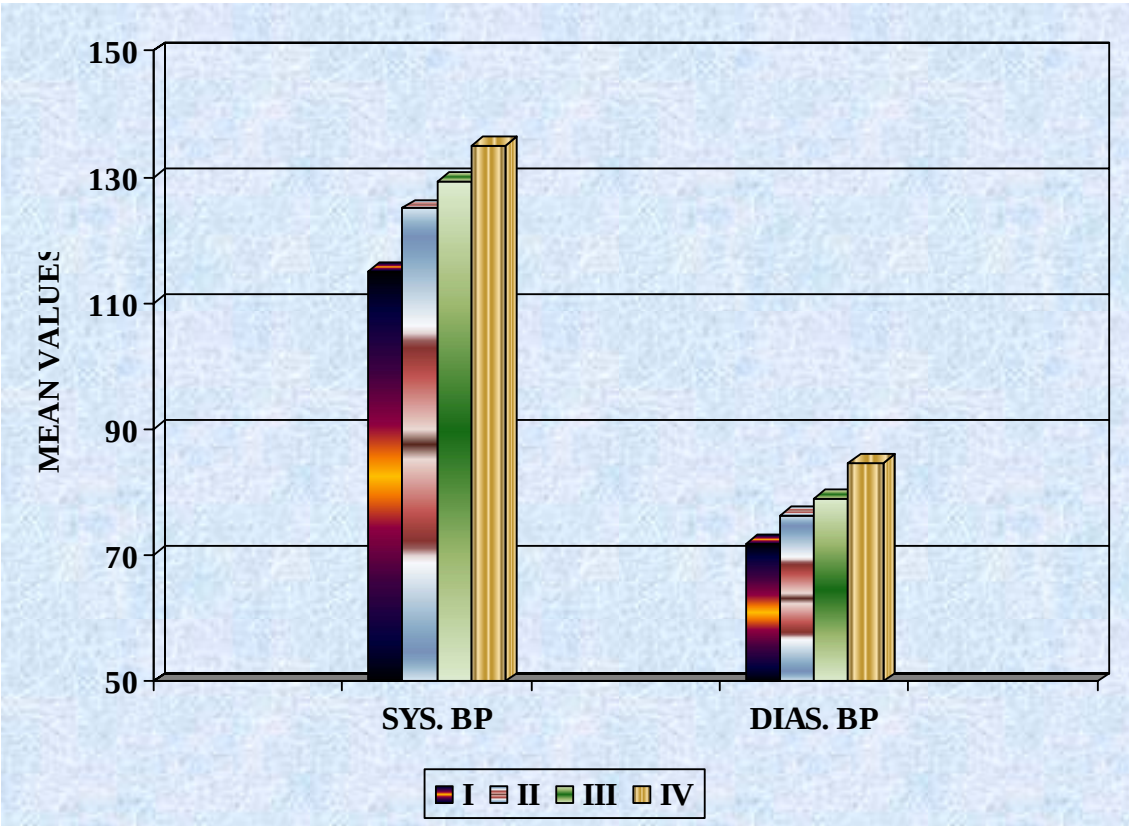
BMI & LIPID PROFILE

FIG:5



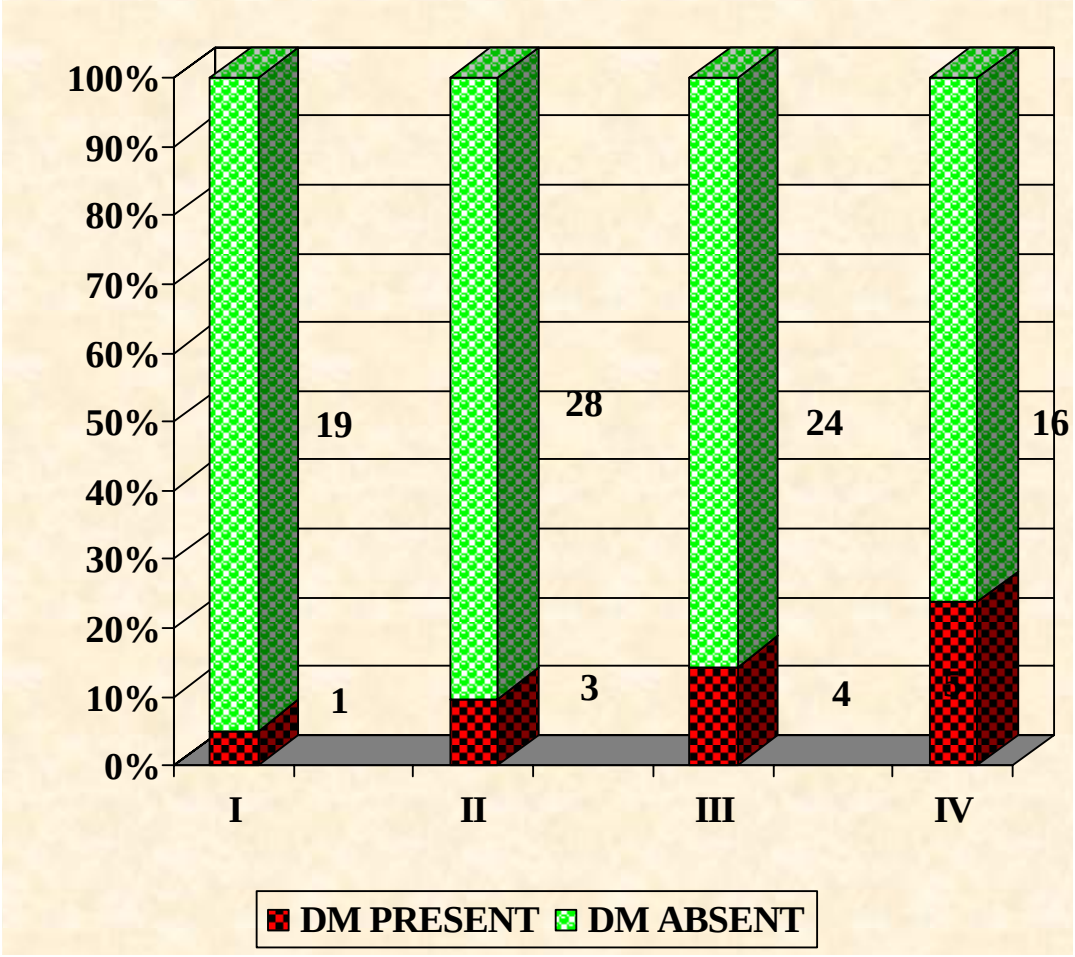
BMI & BLOOD PRESSURE

FIG:4



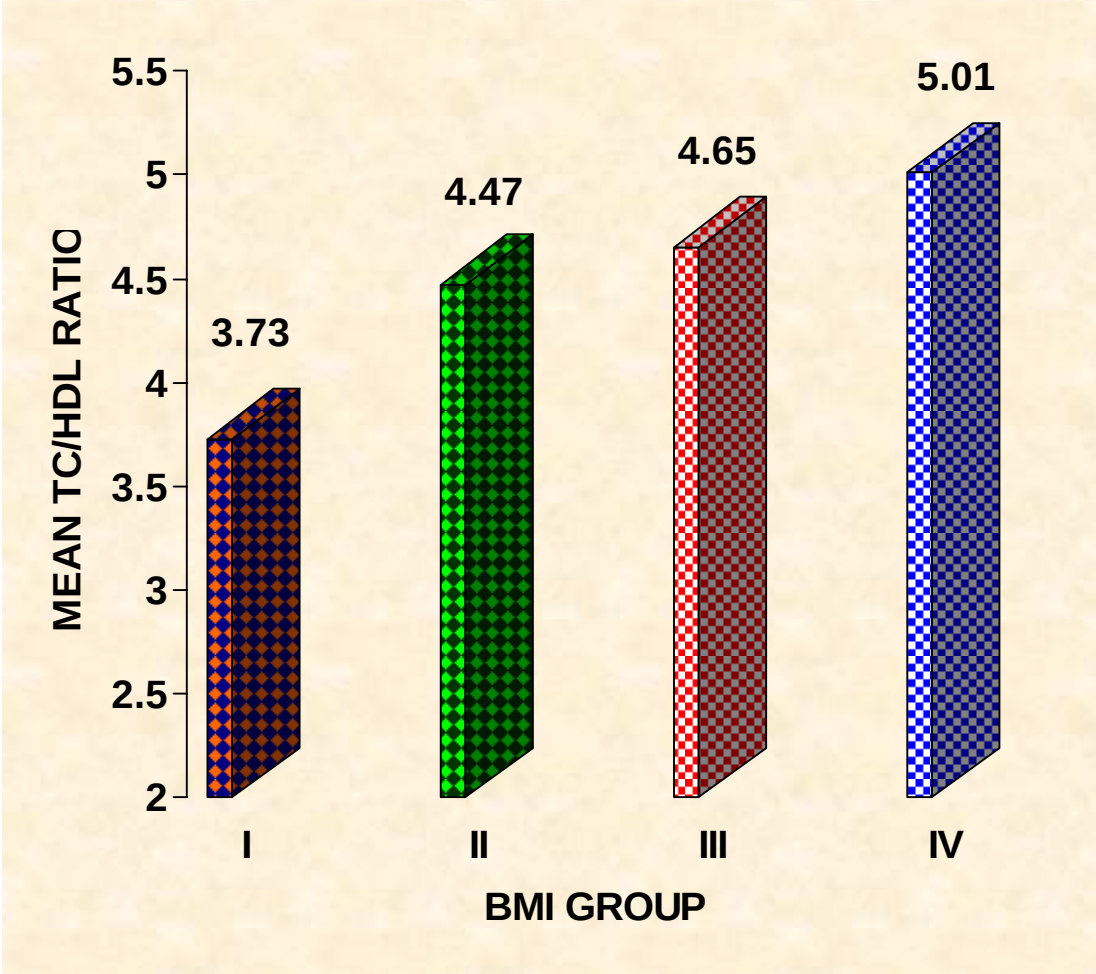
BMI & DM

FIG:9


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
19
1
28
3
24
4
16
I
II
III
IV
DM PRESENT
DM ABSENT

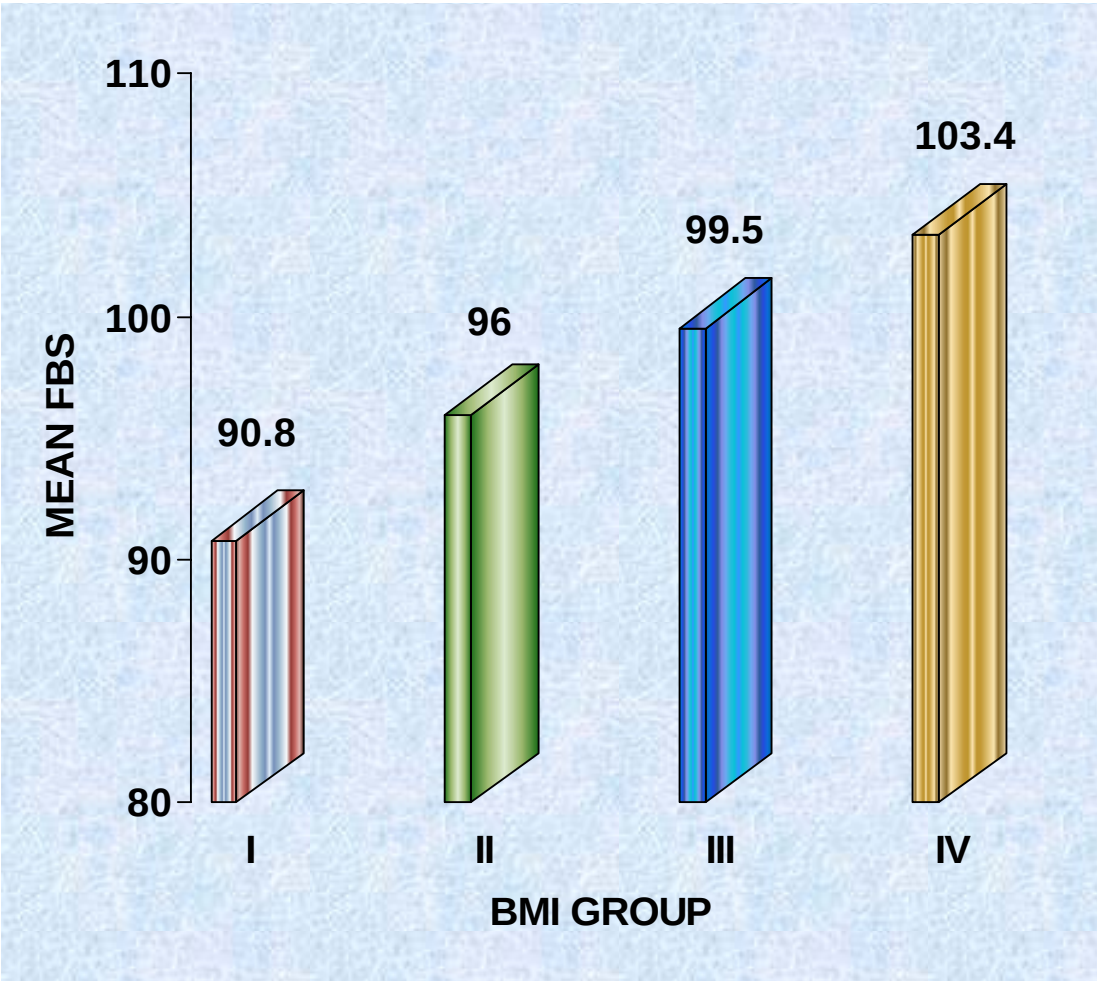
BMI & TC/HDL RATIO

FIG:6



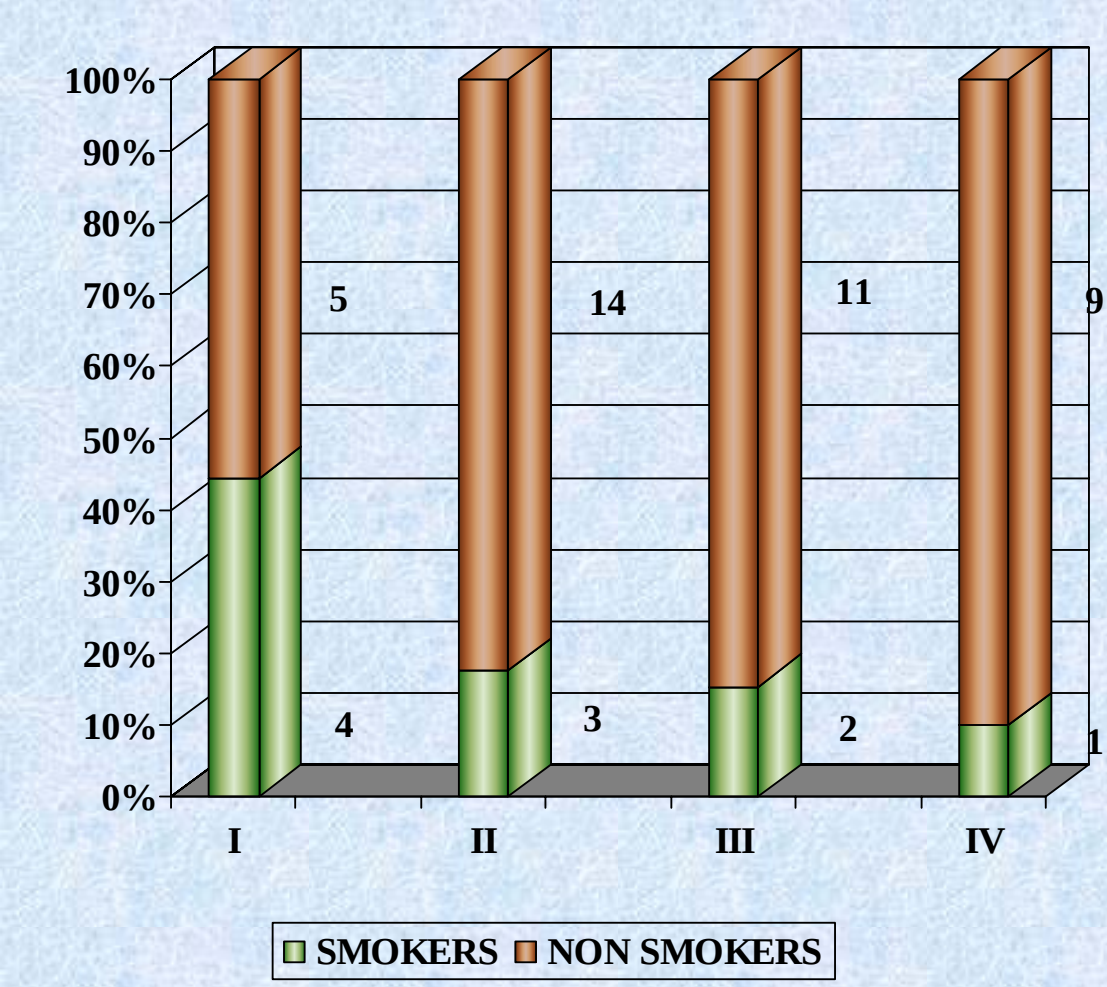
BMI & FASTING BLOOD SUGAR

FIG:8



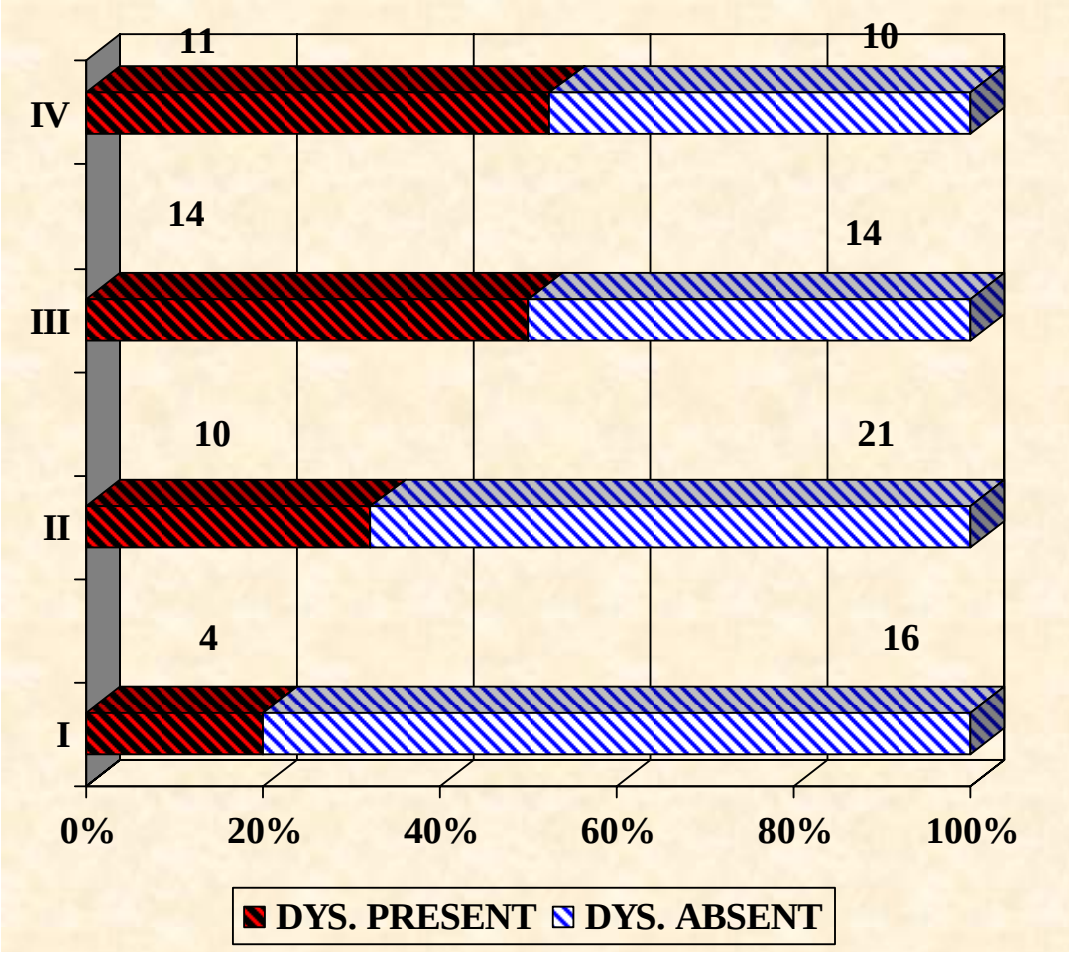
BMI & SMOKING

FIG:10



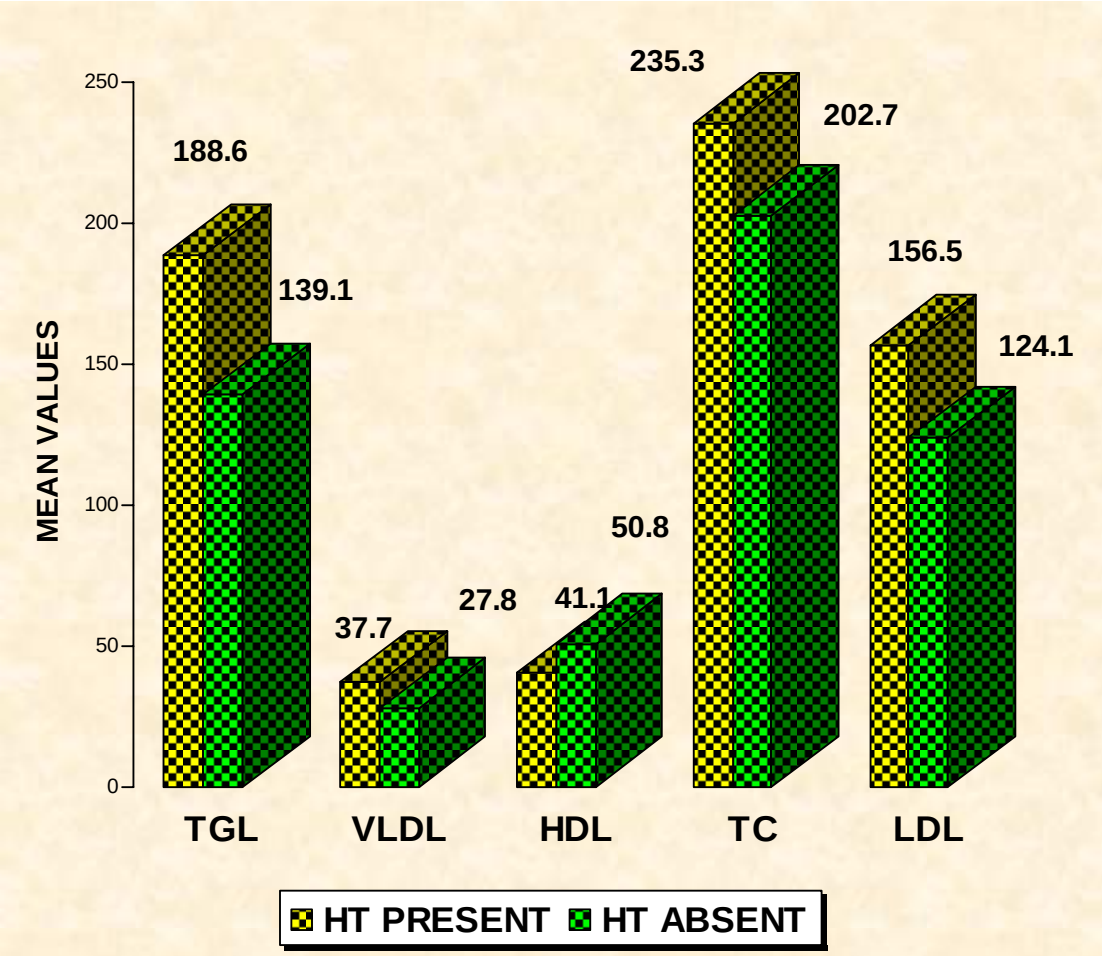
BMI & DYSLIPIDEMIA

FIG:7



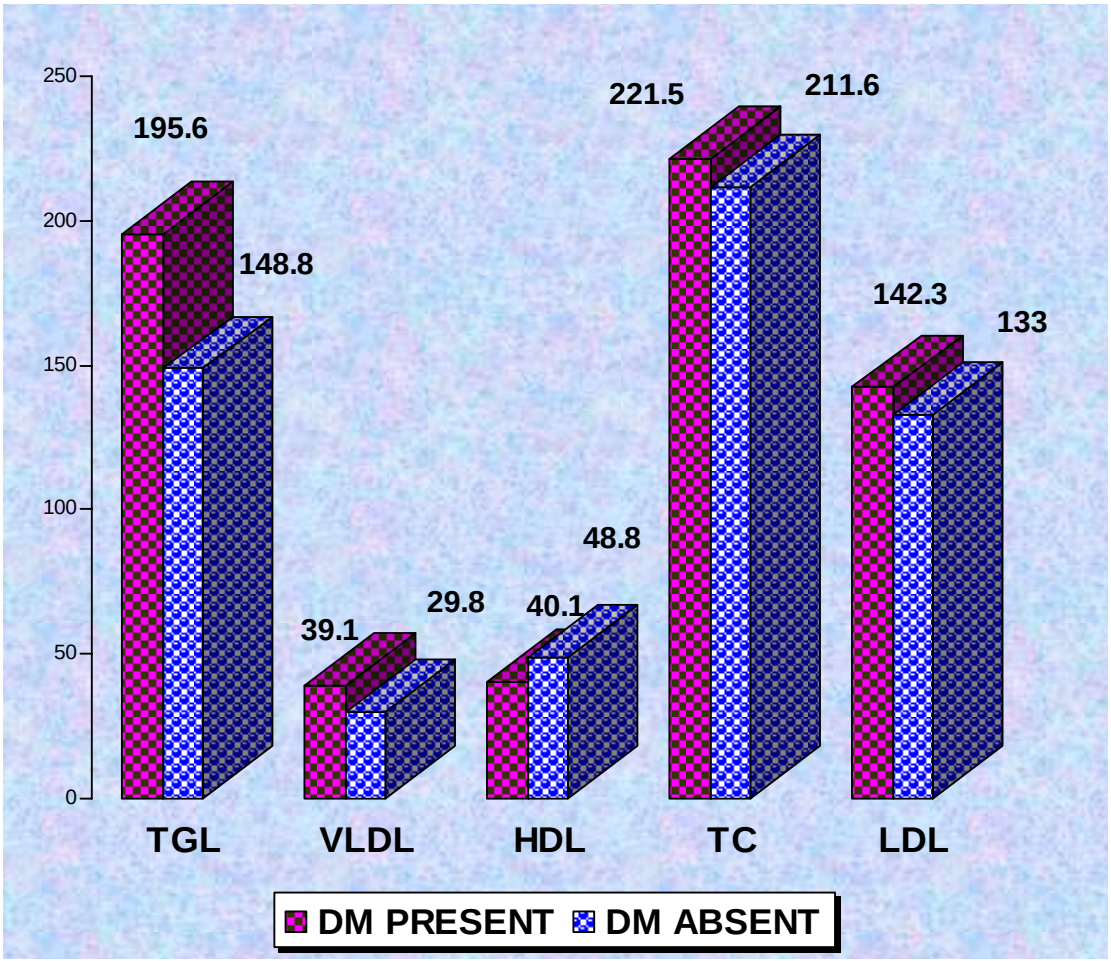
LIPID PROFILE & HT

FIG:13



LIPID PROFILE & DM

FIG:14



Etiology of Obesity

FIG:1

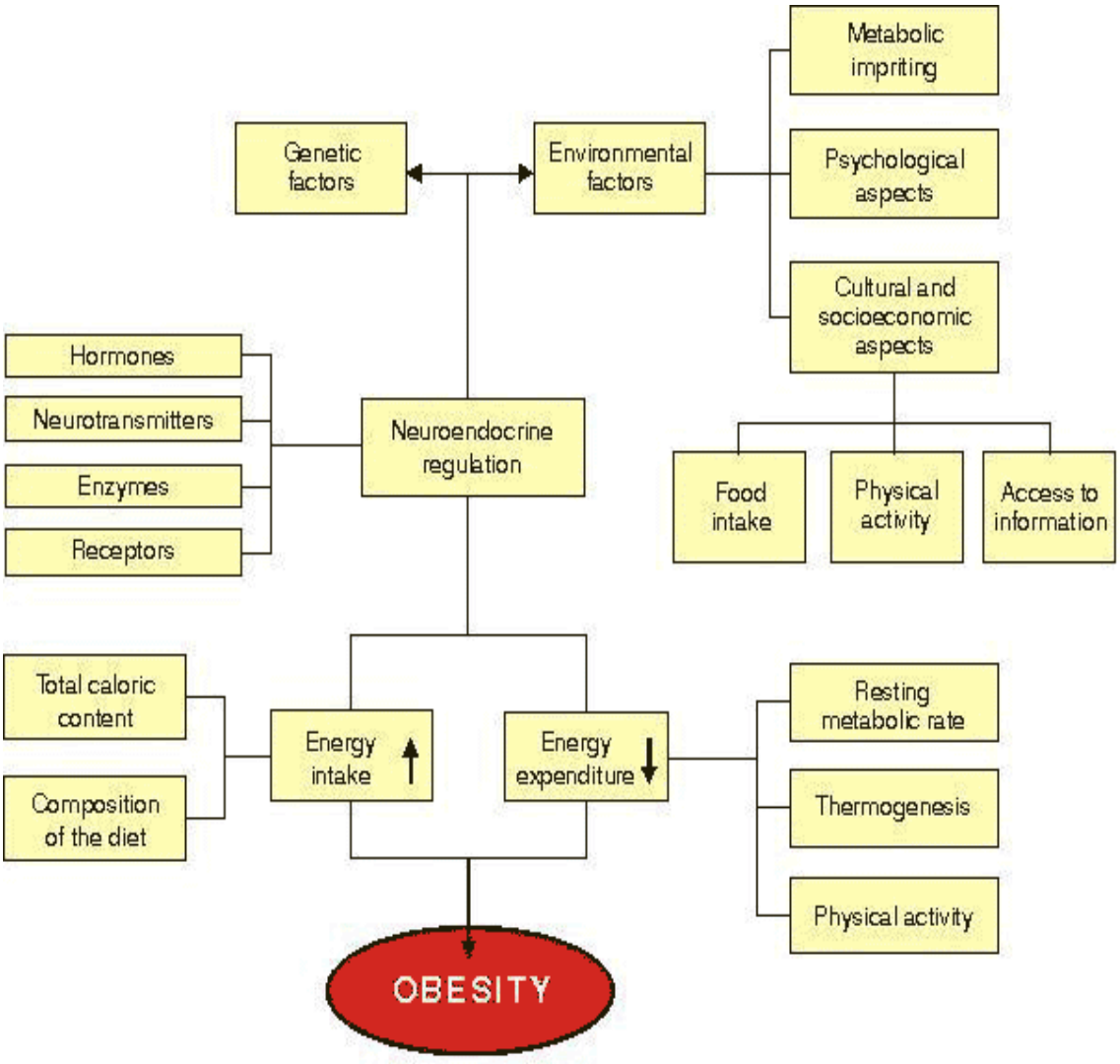


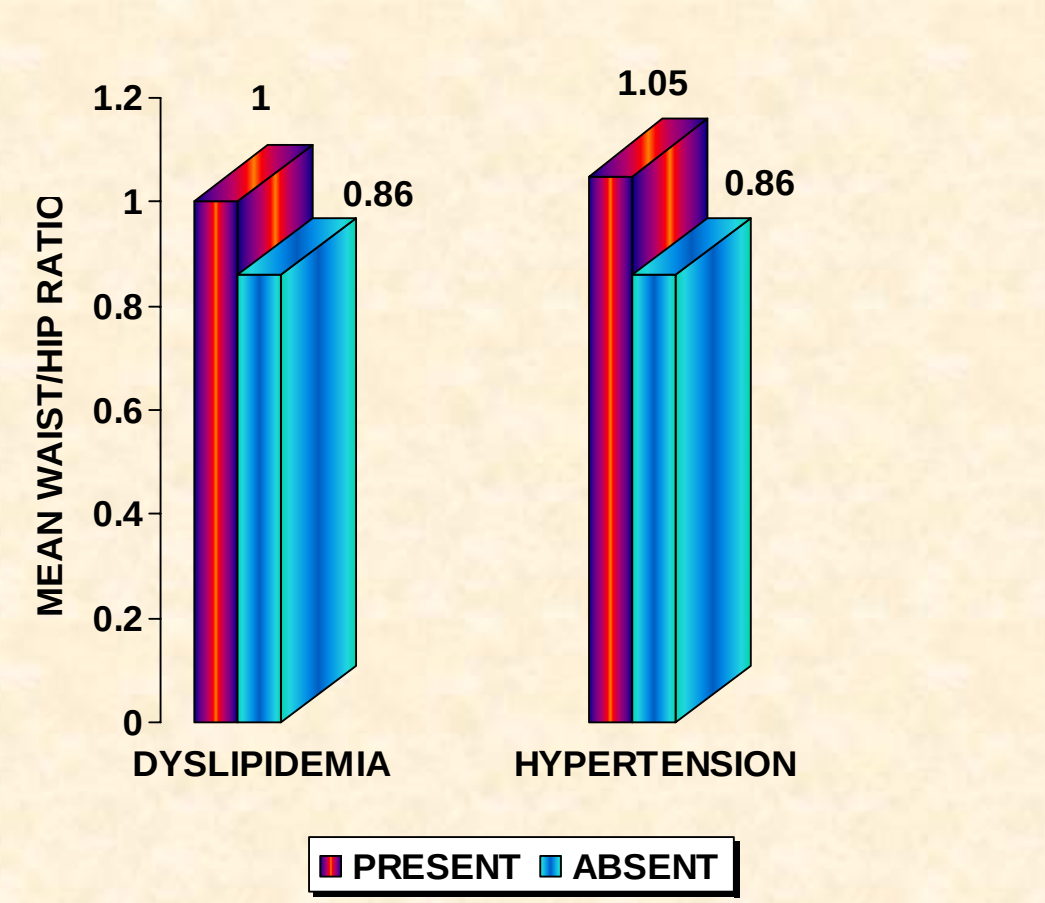
Figure 1 - Causal model for obesity

CORRELATION OF

WAIST /HIP RATIO WITH

DYSLIPIDEMIA AND HYPERTENSION

Fig :12



CORRELATION OF

WAIST CIRCUMFERENCE WITH

DYSLIPIDEMIA AND HYPERTENSION

Fig : 11